(23)

No. 96-188

In The Supreme Court of the United States

October Term, 1996

General Electric Company, *et al.*,
Petitioners,

v.

Robert K. Joiner, *et al.*,
Respondents.

On Writ of Certiorari to the United States Court of Appeals for the Eleventh Circuit

JOINT APPENDIX

John G. Kester *
Steven R. Kuney
Robert J. Shaughnessy
Joanna L. Grossman
Williams & Connolly
725 12th Street, N.W.
Washington, D.C. 20005
(202) 434-5069

Anthony L. Cochran
Chilivis, Cochran, Larkins & Bever
3127 Maple Drive, N.E.
Atlanta, Georgia 30305
(404) 233-4171

Thomas H. Hill
Senior Counsel
Effa G. Farnsworth
Counsel
General Electric Company
3135 Easton Turnpike
Fairfield, Connecticut 06431
(203) 373-3318

Attorneys for Petitioner General Electric Company

Kenneth J. Chesebro *
Robert Godfrey
1600 Massachusetts Avenue
Cambridge, Mass. 02138
(617) 661-4423

Attorneys for Respondents

[Additional Attorneys Continued on Inside Cover]

* Counsel of Record

PETITION FOR CERTIORARI FILED AUGUST 5, 1996
CERTIORARI GRANTED MARCH 17, 1997

525 pp

DAVID H. FLINT
SCHREEDER, WHEELER & FLINT
1600 Candler Building
Atlanta, Georgia 30303
(404) 681-3450
Attorney for Petitioner
Westinghouse Electric
Corporation

EDWARD M. NEWSOM
HAWKINS & PARNELL
4000 SunTrust Plaza
303 Peachtree Street, N.E.
Atlanta, Georgia 30308
(404) 614-7400
Attorney for Petitioner
Monsanto Company

TABLE OF CONTENTS

TABLE OF CONTENTS—Continued

TABLE OF CONTENTS—Continued

NOTICE

The following items have been omitted in printing this appendix because they appear on the following pages in the printed appendix to the petition for certiorari, and are incorporated herein by reference:

Certificates of service wherever appended to documents appearing herein have been omitted in printing.

STATE COURT OF FULTON COUNTY
STATE OF GEORGIA

DOCKET ENTRIES

——CASE——

CASE NO: 92VS0061273 ANNEX: DOWNTOWN
JURISDICTION: ST. CIVIL
CASE TYPE: TORT CASE FILED: 08/05/92
RESTRICTED FILE: AMT $300: YES
JURY DEMAND: YES RUSH PAPERS:

NO. SUMMONS: 03 MRSHL SV GHSE:

JUDGE: JERRY W BAXTER
****SUMMARY DATA** STATUS: ACTIVE**
PLTF: JOINER, ROBERT K
DEFN: GENERAL ELECTRIC COMPANY
DATE JUDGE ASSGN: 08/06/92
TIME JUDGE ASSGN: 1528
NO CLAIMS: 01 NO OPEN CLAIMS: 01
LAST ACTION: ANSWER
DATE: 09/04/92 CONT: NOT CONT.
DISPOSITION:
COLL CLOSE DT: 08/06/92 NO COLL RECS: 01

• • • •

——DEFENDANT——

CASE NO: 92VS0061273 L/D SEQ: 01 NAME:
GENERAL ELECTRIC COMPANY
NAME DESC: A NEW YORK CORPORATION
SHORT NAME: GENERAL EL
ADDRESS: c/o C T CORP SYSTEM
ADDRESS: 1201 PEACHTREE ST STE 1240
CITY: ATLANTA STATE: GA ZIP: 30361
PHONE: BUSINESS PHONE:
COUNSEL: GARY G GRINDLER COUNSEL TYPE:
LIT TYPE: DEFENDANT SOC SEC NO:

CASE NO: 92VS0061273 L/D SEQ: 02 NAME:
WESTINGHOUSE ELECTRIC
NAME DESC: CORPORATION, A PENNSYLVANIA
SHORT NAME: WESTINGHOU
NAME DESC: CORPORATION
ADDRESS: c/o C T CORP SYSTEM
ADDRESS: 1201 PEACHTREE ST STE 1240
CITY: ATLANTA STATE: GA ZIP: 30361
PHONE: BUSINESS PHONE:
COUNSEL: DAVID H FLINT COUNSEL TYPE:
LIT TYPE: DEFENDANT SOC SEC NO:

CASE NO: 92VS0061273 L/D SEQ: 03
NAME: MONSANTO COMPANY
NAME DESC: A DELAWARE CORPORATION
SHORT NAME: MONSANTO C
ADDRESS: c/o PRENTICE HALL CORP SYSTEM
ADDRESS: 66 LUCKIE ST CITY: ATLANTA
STATE: GA ZIP: 30303
PHONE: BUSINESS PHONE:
COUNSEL: JOE C FREEMAN JR COUNSEL TYPE:
LIT TYPE: DEFENDANT SOC SEC NO:

——SERVICE——

CASE NO: 92VS0061273 SEQ NO: 01
DATE SENT: 08/07/92 DATE RECD:

RT/PTNR: 459 SERVICE DATE: 08/07/92
SERVICE TYPE: CORPORATION
LIT TYPE: DEFENDANT
NOTE: WESTINGHOUSE

CASE NO: 92VS0061273 SEQ NO: 02
DATE SENT: 08/10/92 DATE RECD:

RT/PTNR: 446 SERVICE DATE 08/10/92
SERVICE TYPE: CORPORATION
LIT TYPE: DEFENDANT
NOTE: GENERAL ELECTRIC COMPANY

CASE NO: 92VS0061273 SEQ NO: 03
DATE SENT: 08/10/92 DATE RECD:
RT/PTNR: 446 SERVICE DATE: 08/10/92
SERVICE TYPE: CORPORATION
LIT TYPE: DEFENDANT
NOTE: MONSANTO COMPANY

• • • •

——PLAINTIFF——

CASE NO: 9ZVS0061273 SEQ NO: 01
NAME: JOINER, ROBERT K
NAME DESC: SHORT NAME: JOINER, RO
COUNSEL: MICHAEL J WARSHAUER
COUNSEL TYPE:
LIT TYPE: PLTF

——DOCKET ENTRY——

CASE NO: 9ZVS0061273 DATE: 09/04/92
ACTION: ANSWER
LIT TYPE: DEFENDANT L/D SEQ:
EVENT RESULT:
NOTE: OF DEFT MONSANTO COMPANY

CASE NO: 9ZVS0061273 DATE: 09/04/92
ACTION: ANSWER
LIT TYPE: DEFENDANT L/D SEQ:
EVENT RESULT:
NOTE: OF DEFT. GENERAL ELECTRIC CO.

CASE NO: 9ZVS0061273 DATE: 09/04/92
ACTION: ANSWER
LIT TYPE: DEFENDANT L/D SEQ:
EVENT RESULT:
NOTE: OF DEFT. WESTINGHOUSE ELECTRIC CORP.

CASE NO: 9ZVS0061273 DATE: 09/04/92
ACTION: JURY—12 PERSON
LIT TYPE: DEFENDANT L/D SEQ:
EVENT RESULT:
NOTE: OF DEFT. GENERAL ELECTRIC CO

CASE NO: 9ZVS0061278 DATE: 09/04/92
ACTION: MOVE TO US DIS CT
LIT TYPE: DEFENDANT L/D SEQ:
EVENT RESULT:
NOTE: TO U.S. DIST COURT

• • • •

UNITED STATES DISTRICT COURT
FOR THE NORTHERN DISTRICT OF GEORGIA
ATLANTA DIVISION

(Civil Docket for Case #: 92-CV-2137

JOINER, *et al.*

v.

GENERAL ELECTRIC CO. *et al.*

DOCKET ENTRIES

Date	No.	Proceedings
9/4/92	1	NOTICE OF REMOVAL with COMPLAINT.; jury demand (Pretrial instructions attached to service copies and returned to attorney for service.) FILING FEE $120.00 RECEIPT # 178048 (dt) [Entry date 09/09/92]
9/4/92	—	REMOVAL ANSWERS to complaint [1-1] by General Electric Co, Westinghouse Elec, Monsanto Company. Jury demand (dt) [Entry date 09/09/92]
9/10/92	2	Certificate of interested persons. (to judge) (dt) [Entry date 09/11/92]
10/27/92	3	ANSWER TO MANDATORY INTERROGATORIES by pltfs, Robert K. Joiner, Karen P. Joiner (dt) [Entry date 10/29/92]
11/9/92	4	Preliminary Statement and Scheduling Order. (to judge) (dt) [Entry date 11/12/92]
11/19/92	5	Notice of change of address by firm of Freeman & Hawkins. (dt) [Entry date 11/23/92]
11/20/92	6	Preliminary Statement and Scheduling Order. (to judge) (approved by Judge Evans on 12/1/92) (dt) [Entry date 11/24/92] [Edit date 12/04/92]

Date	No.	Proceedings
12/1/92	7	CONSENT ORDER extending time for dfts to respond to mandatory interr thru 12/11/92 by Judge Orinda D. Evans. cc (dt) [Entry date 12/04/92]
12/3/92	8	CONSENT ORDER approved by dep. clerk extending time thru 1/4/93 for dfts to respond to interr etc. cc (dt) [Entry date 12/09/92
12/11/92	9	ANSWER TO MANDATORY INTERROGATORIES by dft Monsanto Company (dt) [Entry date 12/14/92]
12/11/92	10	ANSWER TO MANDATORY INTERROGATORIES by dft Westinghous Elec. (dt) [Entry date 12/14/92]
12/11/92	11	ANSWER TO MANDATORY INTERROGATORIES by dft General Electric Co. (dt) [Entry date 12/14/92]
12/15/92	12	CONSENT ORDER extending time to 1/4/93 by Judge Orinda D. Evans. cc (dt) [Entry date 12/16/92]
1/4/93	13	Stipulation by parties extending time for dfts to respond to interr. thru 1/11/93 (dt) [Entry date 01/07/93]
1/21/93	14	MOTION by plaintiff Robert J. Joiner, plaintiff Karen P. disc. with brief in support. (dt) [Entry date 01/25/93]
1/29/93	15	Notice to take deposition of G. Frame, G. Maiuri, H. Pearce, E. Walsh, R. Barna, J. Aldworth, C. Murray, D. Keiser, R. Kelly, P. Cole, W. Waddell, W. Papageorge by defendant General Electric Co (dt) [Entry date 02/01/93]
2/1/93	16	Response by defendant General Electric Co, defendant Westinghouse Elec, defendant Monsanto Company in support of motion to extend time for disc. by Karen P. Joiner, Robert K. Joiner [14-1] (dt) [Entry date 02/02/93]

Date	No.	Proceedings
2/1/93	17	Application for leave of absence of Joanne Beauvoir Brown w/proposed order. to ct dep. (dt) [Entry date 02/02/93]
2/2/93	—	SUBMITTED on motion to extend time for disc. by Karen P. Joiner, Robert K. Joiner [14-1] to Judge Orinda D. Evans. (dt)
2/3/93	18	ORDER by ct dep. GRANTING leave of absence of Joanne Beauvoir Brown [17-1] for 2/10 to 2/17/93. cc (dt) [Entry date 02/04/93]
2/4/93	19	ORDER GRANTING motion to extend time for disc. by Karen P. Joiner, Robert K. Joiner [14-1] Discovery ends 6/4/93 by Judge Orinda D. Evans. cc (dt) [Entry date 02/05/93]
3/4/93	20	Notice to take deposition of G. Frame by plaintiff Robert K. Joiner, plaintiff Karen P. Joiner (dt) [Entry date 03/05/93]
4/2/93	21	Notice to take deposition of dfts by plaintiff Robert K. Joiner (dt) [Entry date 04/05/93]
4/2/93	22	SUPPLEMENTAL ANSWER TO MANDATORY INTERROGATORIES by Robert K. Joiner, Karen P. Joiner (dt) [Entry date 04/05/93]
6/1/93	23	JOINT REQUEST by parties to extend time for discovery thru 8/3/93. to ODE (bj) [Entry date 06/07/93]
6/10/93	24	CONSENT ORDER GRANTING motion to extend time for discovery thru 8/3/93 by Karen P. Joiner, Robert K. Joiner [21-1] by Judge Orinda D. Evans. cc (dt) [Entry date 06/15/93]
6/30/93	25	Notice to take deposition of D. Huckaby, J. McKinney, W. Mundy, R. Gladish, G. Coulter, R. Lester, G. Bowman by plaintiff Robert K. Joiner, plaintiff Karen P. Joiner (dt) [Entry date 07/01/93]

Date	No.	Proceedings
6/30/93	26	MOTION by plaintiff Robert K. Joiner, plaintiff Karen P. Joiner to extend time for disc. thru 10/31/93 with brief in support. (dt) [Entry date 07/01/93]
6/30/93	27	2nd Supplemental ANSWER TO MANDATORY INTERROGATORIES by pltfs Robert K. Joiner, Karen P. Joiner (dt) [Entry date 07/02/93]
7/6/93	28	MOTION by plaintiff Robert K. Joiner, plaintiff Karen P. Joiner for leave to amend removal complaint [1-1] with brief in support. (dt) [Entry date 07/07/93]
7/8/93	29	Notice to take deposition of Dr. Brown, Dr. Kelly, W. Papageorge by plaintiff Robert K. Joiner, plaintiff Karen P. Joiner (dt) [Entry date 07/09/93]
7/9/93	30	Joint MOTION by parties to extend time for disc. and for scheduling order with brief in support. to ODE (dt) [Entry date 07/13/93]
7/13/93	31	ORDER GRANTING joint motion to extend time for disc. and for scheduling order by Robert K. Joiner [30-1] by Judge Orinda D. Evans. cc (see same for scheduling—disc also stayed pending ruling on any motions in limine or s.j.). (dt) [Entry date 07/14/93]
7/22/93	32	Application for leave of absence of Michael J. Warshauer cc (dt) [Entry date 07/27/93]
7/22/93	33	ORDER by ct dep. GRANTING leave of absence of Michael J. Warshauer [32-1] for 7/29 to 8/16/93. cc (dt) [Entry date 07/27/93]
7/29/93	—	SUBMITTED on Motion for leave to amend removal complaint [1-1] by Karen P. Joiner, Robert K. Joiner [28-1] to Judge Orinda D. Evans. (dt)

Date	No.	Proceedings
8/4/93	34	ORDER GRANTING motion for leave to amend removal complaint [1-1] by Karen P. Joiner, Robert K. Joiner [28-1] by Judge Orinda D. Evans. cc (dt) [Entry date 08/05/93]
8/20/93	35	Stipulation by parties extending time thru 9/10/93 for dfts to respond to disc. being pltf's 2nd interr etc (dt) [Entry date 02/23/93]
8/24/93	36	AMENDED COMPLAINT [1-1] by Robert K. Joiner, Karen P. Joiner; jury demand. (dt) [Entry date 08/25/93]
9/1/93	37	EXPERT WITNESS designation by defendant Monsanto Company, defendant General Electric Co, defendant Westinghouse Elec (dt) [Entry date 09/02/93]
9/2/93	38	ANSWER to amended complaint [36-1] by Westinghouse Elec (dt) [Entry date 09/03/93]
9/2/93	39	ANSWER to amended complaint [36-1] by Monsanto Company (dt) [Entry date 09/03/93]
9/3/93	40	ANSWER to amended complaint [36-1] by General Electric Co (dt) [Entry date 09/07/93]
9/30/93	41	SECOND Notice to take deposition of W. Waddell, P. Cole by plaintiff Robert K. Joiner, plaintiff Karen P. Joiner (dt) [Entry date 10/01/93]
10/12/93	42	Notice of change of address by firm of Burge & Wettermark. . (dt) [Entry date 10/13/93]
10/19/93	43	SUPPLEMENTAL ANSWER TO MANDATORY INTERROGATORIES by dft Monsanto Company (dt) [Entry date 10/20/93]
10/21/93	44	Second Supplemental ANSWER TO MANDATORY INTERROGATORIES by dft Monsanto Company (dt) [Entry date 10/25/93]

Date	No.	Proceedings
12/7/93	45	CONSENT ORDER extending time for dfts to file motion for s.j thru 12/8/93 by Judge Orinda D. Evans. cc (dt) [Entry date 12/08/93]
12/8/93	46	JOINT MOTION by defendant General Electric Co, defendant Westinghouse Elec, defendant Monsanto Company for summary judgment and statement of material facts with brief in support. (dt) [Entry date 12/10/93]
12/8/93	47	MOTION (request) by defendant General Electric Co., defendant Westinghouse Elec, defendant Monsanto Company for oral argument on motion for s.j. with brief in support. (dt) [Entry date 12/10/93]
12/8/93	48	Notice of filing disc. in support of s.j. by defendant General Electric Co, defendant Westinghouse Elec, defendant Monsanto Company. (dt) [Entry date 12/10/93]
12/8/93	—	Deposition of T. Rouse, R. Joiner, D. Teitelbaum, L. Robertson, G. Frame, J. Brown taken in regards to s.j. for defendant General Electric Co, defendant Westinghouse Elec, defendant Monsanto Company (dt) [Entry date 12/10/93]
12/8/93	49	REQUEST for filing disc. material by defendant General Electric Co., defendant Westinghouse Elec, defendant Monsanto Company (dt) [Entry date 12/10/93]
12/10/93	—	Notice of motion for summary judgment, statement of material facts by Monsanto Company, Westinghouse Elec, General Electric Co [46-1] filed 12/8/93, mailed 12/10/93. (dt)
12/28/93	50	MOTION by plaintiff Robert K. Joiner, plaintiff Karen P. Joiner to extend time to 1/31/94 to respond to motion for summary judgment and statement of material facts by Monsanto Company, Westinghouse Elec, General Electric Co [46-1] with brief in support. (dt) [Entry date 01/03/94]

Date	No.	Proceedings
1/3/94	51	CONSENT ORDER approved by dep. clerk extending time for 1 week for pltf to respond to motion for summary judgment and statement of material facts by Company, Westinghouse Elec, General Electric Co [46-1]. cc (dt) [Entry date 01/05/94]
1/5/94	52	CONSENT ORDER GRANTING motion to extend time to 1/31/94 to respond to motion for summary judgment and statement of material facts by Monsanto Company, Westinghouse Elec, General Electric Co [46-1] by Karen P. Joiner, Robert K. Joiner [50-1] by Judge Orinda D. Evans. cc (dt) [Entry date 01/06/94]
1/31/94	53	Response by plaintiff Robert K. Joiner, plaintiff Karen J. Joiner w/affidavit in opposition to motion for summary judgment and Response to statement of material facts by Monsanto Company, Westinghouse Elec, General Electric Co [46-1] (dt) [Entry date 02/02/94]
1/31/94	54	REQUEST by plaintiff Robert K. Joiner, plaintiff Karen P. Joiner for dfts to file orig. disc. (dt) [Entry date 02/02/94]
1/31/94	55	MOTION (request) by plaintiff Robert K. Joiner, plaintiff Karen P. Joiner for oral argument on motion for s.j. [Entry date 02/02/94]
1/31/94	56	Notice of filing orig. disc. by plaintiff Robert K. Joiner, plaintiff Karen P. Joiner (dt) [Entry date 02/02/94]
1/31/94	—	Deposition of A. Schecter, W. Bailey, P. Cole, W. Waddell, S. Hamilton, W. Papageorge and 2 vol of medical records of pltf. Robert Joiner taken for plaintiff Robert K. Joiner, plaintiff Karen P. Joiner in regards to s.j. (dt) [Entry date 02/02/94]

Date	No.	Proceedings
2/7/94	—	SUBMITTED on motion for summary judgment and statement of material facts by Monsanto Company, Westinghouse Elec, General Electric Co [46-1], motion for oral argument on motion for s.j. by Monsanto Company, Westinghouse Elec, General Electric Co [47-1], motion for oral argument on motion for s.j. by Karen P. Joiner, Robert K. Joiner to Judge Orinda D. Evans (dt)
2/28/94	57	Joint Memorandum by dfts in REPLY to response to motion for summary judgment [46-1] w/appendix. (dt) [Entry date 03/03/94] [Edit date 03/03/94]
3/16/94	58	Notice of filing disc. material by dfts (dt) [Entry date 03/18/94]
6/16/94	59	MOTION by deft GE for leave to file supplemental brief [Entry date 6/20/94]
7/14/94	60	ORDER GRANTING motion for leave to file supplemental brief by General Electric Co [59-1] by Judge Orinda D. Evans. cc sent out by ct dep. (dt) [Entry date 07/21/94]
7/19/94	—	SUBMITTED on motion for leave to file supplemental brief by General Electric Co [59-1] to Judge Orinda D. Evans. (dt)
7/22/94	61	Application for leave of absence of Michael J. Warshauer (dt) [Entry date 07/25/94]
7/22/94	62	ORDER by ct dep. GRANTING leave of absence of Michael J. Warshauer [61-1] for 7/29 to 8/15/94. cc (dt) [Entry date 07/25/94]
7/25/94	63	Supplemental brief by dft General Electric to motion for summary judgment by dfts [46-1] (dt) [Entry date 08/02/94]
8/8/94	64	MOTION by pltfs for leave to file brief in response to GE's Suppl. brief (dt) [Entry date 08/10/94]

Date	No.	Proceedings
8/15/94	65	ORDER GRANTING motion for leave to file brief in response to GE's Suppl. brief by Robert K. Joiner [64-1] by Judge Orinda D. Evans. cc (dt) [Entry date 02/18/94]
8/29/94	66	Reply brief to supplemental brief by General Electric Co [63-1] by pltfs. (dt) [Entry date 08/30/94]
9/1/94	67	MOTION by dft General Electric Co for leave to file supplemental brief (dt) [Entry date 09/07/94]
9/13/94	68	Response by pltfs in opposition to motion for leave to file supplemental brief by General Electric Co [67-1] (dt) [Entry date 09/14/94]
9/16/94	69	ORDER GRANTING motion for summary judgment by Monsanto, Westinghouse, General Electric [46-1], DENYING motion for oral argument on motion for s.j. by Monsanto Company, Westinghouse Elec, General Electric Co [47-1], DENYING motion for oral on motion for s.j. by pltfs. [55-1], DENYING motion for leave to file supplemental brief by General Electric Co [67-1] by Judge Orinda D. Evans. cc (dt) [Entry date 09/20/94]
9/16/94	70	JUDGMENT ENTERED on amend/amended complaint [36-1], removal complaint [1-1] for dfts againts pltfs for costs of action. cc (dt) [Entry date 09/20/94]
9/27/94	71	NOTICE OF APPEAL from order [69-1], judgment. copy of dkt, NOA, order and judgment to USCA. Letter sent to counsel to pay fee. (dt) [Entry date 10/05/94]
10/13/94	—	USCA appeal fees received $105.00. r/209923. USCA notified (dt)
10/14/94	72	Bill of costs of defendant Westinghouse Elec. (dt) [Entry date 10/18/94]

Date	No.	Proceedings
10/14/94	73	Bill of costs of defendant General Electric Co. (dt) [Entry date 10/18/94]
10/14/94	74	Bill of costs of defendant Monsanto Company. (dt) [Entry date 10/18/94]
10/18/94	—	COSTS TAXED in the amount of $4,067.98 for defendant Westinghouse Elec against pla. (dt)
10/18/94	—	COSTS TAXED in the amount of $4,684.72 for defendant General Electric Co against pla. (dt)
10/18/94	—	COSTS TAXED in the amount of $4,548.74 for defendant Monsanto Company against pla. (dt)
10/18/94	—	Acknowledgment by Court of Appeals regarding appeal by Karen P. Joiner, Robert K. Joiner [71-1]. USCA dkt no. 94-9131 (jps) [Entry date 10/19/94]
11/1/94	—	Request for Certificate of Readiness from Court of Appeals regarding appeal. (fem) [Entry date 11/02/94]
11/9/94	—	CERTIFICATE OF READINESS OF RECORD ON APPEAL with certified copy of docket mailed to USCA. (5 vol. pleadings; 12 vol. depositions; 2 vol. medical records). (fem)
11/22/94	—	USCA acknowledges receipt of COR. (fem)
2/24/95	—	FORTHWITH LETTER from U.S. Court of Appeals regarding appeal. (oug) [Entry date 03/07/95]
2/28/95	—	Certified and TRANSMITTED RECORD to U.S. Court of Appeals appeal by Karen P. Joiner, Robert K. Joiner [71-1]. (5 vol. of pleadings, 12 depos. and 2 vols. of medical records). (oug) [Entry date 03/07/95]

Date	No.	Proceedings
6/12/96	75	Certified copy of ORDER of the U.S. Court of Appeals DENYING appellees petition for rehearing. cc (dt) [Entry date 6/20/96]
7/11/96	76	Certified copy of ORDER of the U.S. Court of Appeals GRANTING G.E.'s motion to stay mandate pending petition for writ of certiorari, cc (dt) [Entry date 07/12/96]
8/5/96	77	Certified copy of ORDER of the U.S. Court of Appeals GRANTING appellees' motion for ext. of time to stay issuance of mandate to 8/5/96. cc (dt) [Entry date 08/06/96]

UNITED STATES COURT OF APPEALS
FOR THE ELEVENTH CIRCUIT

No. 94-9131

ROBERT K. JOINER, KAREN P. JOINER,
Plaintiffs-Appellants,

versus

GENERAL ELECTRIC COMPANY,
A New York Corporation;
WESTINGHOUSE ELECTRIC CORPORATION,
A Pennsylvania Corporation;
MONSANTO COMPANY,
A Delaware Corporation,
Defendants-Appellees.

DOCKET ENTRIES

Date	Proceedings
11/29/94	Civil Docketing Statement
10/17/94	Staff Atty. Jurisdiction
10/21/94	Probable Jurisdiction Noted
11/10/94	Certificate of Readiness due 11/18/94
3/1/95	ROA (# Vols. 5, p. 5, T —) due 1/06/95
3/1/95	Exhibits (Itemize) 2 boxes Depositions, Exhibits
	BRIEFING INFORMATION
1/17/95	Brief for Appellant D: 1/13 due 1/13/95
1/13/95	Record Excerpts
2/21/95	Brief for Appellee M3/1 D:2/21 due 2-20-95

Date	Proceedings
3/13/95	Reply Brief for Appellants 3-10-95
6/13/95	Supp. Authority Apt — Ape x
6/22/95	Supp. Authority Apt x Ape — CE'd to Panel
6/30/95	Supp. Auth. Ape x CE'd to panel
	OTHER MOTIONS & ORDERS
4/29/96	Flg. motion of appellant to file bill of costs out of time. dc
5/24/96	FLG. ORDER: Appellant's motion to file bill of costs out of time is GRANTED. (RB) (J) smg
6/18/96	Flg. Appellees' motion for stay of issuance of mandate. dc
6/20/96	Flg. response letter in opposition to stay more than 30 days. dc
7/9/96	Flg. ORDER that Motion of General Electric to Stay mandate pending certiorari to and including 7/18/96 is GRANTED/RB/. dc
7/15/96	Flg. unopposed motion to extend issuance of mandate pending certiorari. dc
8/2/96	Flg. ORDER that Motion of Appellees' for extension of time to stay the issuance of mandate to and including 8/5/96 is GRANTED/RB/. dc
	CALENDAR INFORMATION
4/2/95	Argument Scheduled for 6/13/95 in ATLANTA #339
	Argument Panel SFB/RB/SMITH
6/13/95	Case Argued—By Apt x By Ape x
	OPINION INFORMATION
3/27/96	Opinion issued
	RB signed SFB conc. spec. RE Smith dissent

Date	Proceedings
	JUDGMENT AND MANDATE INFORMATION
6/3/96	Bill of Costs Filed—Costs Awarded To
8/27/96	Judgment Entered
7/9/96	Mandate Stayed to 7/18/96/*8/5/96
	REHEARING INFORMATION
4/17/96	Petition for Rehearing M J
	Ape Panel En Banc
6/11/96	Order Denying Rehearing
	SUPREME COURT INFORMATION—NO. 96-188
8/9/96	Notice of Flg. Cert. on 8/5/96
12/5/96	Cert. Record Transmitted 5 vols/2 Bx-Exh.

IN THE STATE COURT OF FULTON
STATE OF GEORGIA

JURY TRIAL DEMANDED

Civil Action File No. 92-VS-61273A

ROBERT K. JOINER, and KAREN P. JOINER,
Plaintiffs,

v.

GENERAL ELECTRIC COMPANY,
a New York Corporation,
WESTINGHOUSE ELECTRIC CORPORATION,
a Pennsylvania Corporation,
and MONSANTO COMPANY,
a Delaware Corporation,
Defendants.

COMPLAINT

NOW COME Plaintiffs, and file this Complaint against Defendants as follows:

1.

Defendant, General Electric Company, a New York corporation, ("GE") maintains its registered agent in Fulton County, Georgia, committed a tortious act in Georgia, and is subject to the jurisdiction of the courts of this state, venue in this Court is proper, and it may be served with Summons and Complaint as allowed by law.

2.

Defendant, Westinghouse Electric Corporation, a Pennsylvania corporation ("Westinghouse") maintains its registered agent in Fulton County, Georgia, committed a tortious act in Georgia, and is subject to the jurisdiction of the courts of this state, venue in this Court is proper,

and it may be served with Summons and Complaint as allowed by law.

3.

Defendant, Monsanto Company, a Delaware corporation, ("Monsanto"), maintains its registered agent in Fulton County, Georgia, committed a tortious act in Georgia, and is subject to the jurisdiction of the courts of this state, venue in this Court is proper, and it may be served with Summons and Complaint as allowed by law.

4.

Defendant Monsanto manufactured Polychlorinated Biphenyls ("PCB") to be used in the manufacturing, testing and as a cooling fluid in electrical transformers manufactured by Defendants GE and Westinghouse.

5.

Defendants GE and Westinghouse manufactured and sold as new products electrical transformers containing PCB fluid manufactured by Defendant Monsanto.

6.

At the time of the manufacturing of these transformers with PCB fluid used in the manufacturing, testing and ultimate product, all Defendants were aware that PCBs were unreasonably dangerous and likely to cause injury and death to human beings who were exposed to the PCB fluids, including PCB residues in mineral oil fluids, during service and use of the transformer products.

7.

Plaintiff Robert K. Joiner was an ultimate, foreseeable, intended user and servicer of the transformer products manufactured by Defendants GE and Westinghouse which contained PCB fluids, and PCB contamination, manufactured by Defendant Monsanto.

8.

Throughout the course of his use of the transformers in his capacity as a repairman, Plaintiff Robert K. Joiner was caused to be exposed to PCB, was caused to be exposed to PCB fumes, and was caused to inhale PCB contaminated fluids, mists, liquids, and steams and he was also caused to absorb PCB through contact with his skin.

9.

As a result of Plaintiff Robert K. Joiner's exposure to PCBs, he contracted terminal lung cancer.

COUNT ONE
(STRICT LIABILITY AGAINST DEFENDANTS GE AND WESTINGHOUSE)

10.

Plaintiffs repeat, reallege, and incorporate by reference, the allegations of paragraphs 1 through 9 as if repeated herein as paragraph 10.

11.

Defendants put an unreasonably dangerous and defective product, the PCB contaminated electrical transformers, into the stream of commerce. The defect was the proximate cause of a serious bodily injury sustained by Plaintiff Robert K. Joiner. As a result, Defendants are strictly liable to Plaintiff.

12.

As a direct and proximate result of Defendants' acts, Plaintiff Robert K. Joiner was injured and suffered damages including permanent physical disfigurement, permanent physical disability, past pain and suffering, future pain and suffering, past medical expenses and lost wages, and future medical expenses and lost wages, all entitling him to an award of damages in his favor and against Defendants, and each of them, jointly and

severally, in an amount in excess of Ten Million Dollars ($10,000,000.00.)

COUNT TWO
(NEGLIGENCE AGAINST DEFENDANTS GE AND WESTINGHOUSE)

13.

Plaintiff repeats, realleges, and incorporates by reference, the allegations of paragraphs 1 through 12 as if repeated herein as paragraph 13.

14.

Defendants GE and Westinghouse negligently manufactured and distributed the electrical transformers in that: (i) the electrical transformers were sold in a condition which allowed it to be contaminated with PCBs and cause serious injury in expected use; (ii) the electrical transformers were defective in that they were manufactured and sold with PCB contamination which was likely to cause injury in the use for which they were intended; and (iii) the electrical transformers were sold without adequate warnings and instructions with respect to the known and unknown dangers of PCBs contained within them. This negligence was the proximate cause of a serious bodily injury sustained by Plaintiff.

15.

As a direct and proximate result of Defendant GE's and Westinghouse's negligence, Plaintiff was injured and has suffered damages including permanent physical disfigurement, permanent physical disability, past pain and suffering, future pain and suffering, past medical expenses and lost wages, and future medical expenses and lost wages, all entitling him to an award of damages in his favor and against Defendants, and each of them, jointly and severally, in an amount in excess of Ten Million Dollars ($10,000,000.00.)

COUNT THREE
(FRAUD AGAINST DEFENDANTS GE AND WESTINGHOUSE)

16.

Plaintiff repeats, realleges, and incorporates by reference, the allegations of paragraphs 1 through 15 as if repeated herein as paragraph 16.

17.

Defendants GE and Westinghouse represented to the buying public and those who serviced their electrical transformer products, including Plaintiff Robert K. Joiner, that the electrical transformer products were free from PCBs and were safe in regular use and servicing.

18.

Defendants GE and Westinghouse intended for the buying public and those who serviced their electrical transformer products, including Plaintiff Robert K. Joiner, to rely on their representations that the electrical transformer products were free from PCB and were safe in regular use and servicing.

19.

The buying public and those who serviced their electrical transformer products, including and particularly Plaintiff Robert K. Joiner, reasonably relied on the representations of Defendants GE and Westinghouse that the electrical transformer products were free from PCBs and were safe in regular use and servicing and as a result Plaintiff Robert K. Joiner took no precautions to protect himself when handling and servicing the electrical transformer products.

20.

As a direct and proximate result of the justifiable reliance on Defendant GE's and Westinghouse's fraudulent misrepresentations, Plaintiff Robert K. Joiner was exposed

to PCBs and was caused to contract terminal lung cancer and was otherwise injured and has suffered damages including permanent physical disfigurement, permanent physical disability, past pain and suffering, future pain and suffering, past medical expenses and lost wages, and future medical expenses and lost wages, all entitling him to an award of damages in his favor and against Defendants, and each of them, jointly and severally, in an amount in excess of Ten Million Dollars ($10,000,000.00.)

COUNT FOUR
(STRICT LIABILITY AGAINST DEFENDANT MONSANTO)

21.

Plaintiffs repeat, reallege, and incorporate by reference, the allegations of paragraphs 1 through 20 as if repeated herein as paragraph 21.

22.

Defendant Monsanto put an unreasonably dangerous and defective product, the PCB fluids, into the stream of commerce. The defect was the proximate cause of a serious bodily injury sustained by Plaintiff Robert K. Joiner. As a result, Defendants are strictly liable to Plaintiff.

23.

As a direct and proximate result of Defendant Monsanto's acts, Plaintiff Robert K. Joiner was injured and suffered damages including permanent physical disfigurement, permanent physical disability, past pain and suffering, future pain and suffering, past medical expenses and lost wages, and future medical expenses and lost wages, all entitling him to an award of damages in his favor and against Defendants, and each of them, jointly and severally, in an amount in excess of Ten Million Dollars ($10,000,000.00.)

COUNT FIVE
(NEGLIGENCE AGAINST DEFENDANT MONSANTO)

24.

Plaintiff repeats, realleges, and incorporates by reference, the allegations of paragraphs 1 through 23 as if repeated herein as paragraph 24.

25.

Defendant Monsanto negligently manufactured and distributed the PCB fluid in that: (i) the PCB fluid was unreasonably dangerous and known to be likely to cause serious injury in expected use; (ii) the PCB fluid was sold without adequate warnings and instructions with respect to the known and unknown dangers of PCBs. This negligence was the proximate cause of a serious bodily injury sustained by Plaintiff.

26.

As a direct and proximate result of Defendant Monsanto's negligence, Plaintiff was injured and has suffered damages including permanent physical disfigurement, permanent physical disability, past pain and suffering, future pain and suffering, past medical expenses and lost wages, and future medical expenses and lost wages, all entitling him to an award of damages in his favor and against Defendants, and each of them, jointly and severally, in an amount in excess of Ten Million Dollars ($10,000,000.00.).

COUNT SIX
(LOSS OF CONSORTIUM AGAINST ALL DEFENDANTS)

27.

Plaintiff repeats, realleges, and incorporates by reference, the allegations of paragraphs 1 through 26 as if repeated herein as paragraph 27.

28.

At all times relevant to this action, Plaintiff Karen P. Joiner has been the wife of Plaintiff Robert K. Joiner.

29.

As a direct and proximate result of the wrongful conduct of Defendants, and each of them, Plaintiff Robert K. Joiner suffered severe and permanent bodily injuries including serious and permanent physical and emotional injury and disability, substantial and continuing pain, suffering, and discomfort, and lost wages and medical expenses, both past and future. As a result of these injuries to her husband, Plaintiff Karen P. Joiner has lost the domestic and conjugal services previously provided to her by her husband and has been deprived of her right to consortium.

30.

As a direct and proximate result of Defendants' wrongful conduct, Plaintiff Karen P. Joiner is entitled to an award of general damages for her loss of consortium in an amount to be determined by a jury of her peers in an amount in excess of Ten Million Dollars ($10,000,000.00.) against each of them, jointly and severally.

COUNT SEVEN
(PUNITIVE DAMAGES AGAINST ALL DEFENDANTS)

31.

Plaintiff repeats, realleges, and incorporates by reference, the allegations of paragraphs 1 through 30 as if repeated herein as paragraph 31.

32.

Defendants' conduct in defrauding Plaintiff is intentional, in bad faith, and in wanton disregard of Plaintiff's rights. Defendants' conduct in distributing and selling

defective and dangerous products which Defendants knew were likely to cause bodily injury was willful, wanton, and in callous disregard for human health and safety.

33.

As a direct and proximate result of Defendants' wrongful conduct, Plaintiffs are entitled to judgment against Defendants, and each of them, jointly and severally, for exemplary damages to deter each of them from similar conduct in the future and to punish them for their wrongful acts in an amount to be determined by the enlightened conscience of the finder of fact in an amount in excess of Twenty Million Dollars ($20,000,000.00).

WHEREFORE, Plaintiff prays that summons issue, that Defendants be served and made to appear and answer, that a JURY TRIAL be held, and that they be awarded judgment in their favor and against the Defendants, jointly and severally, as follows:

(a) Under Count One, that Plaintiff Robert K. Joiner be awarded his special damages as are proved at trial plus an appropriate amount as general damages in an amount in excess of Ten Million Dollars ($10,000,000.00.);

(b) Under Count Two, that Plaintiff Robert K. Joiner be awarded his special damages as are proved at trial plus an appropriate amount as general damages in an amount in excess of Ten Million Dollars ($10,000,000.00.);

(c) Under Count Three, that Plaintiff Robert K. Joiner be awarded his special damages as are proved at trial plus an appropriate amount as general damages in an amount in excess of Ten Million Dollars ($10,000,000.00.);

(d) Under Count Four, that Plaintiff Robert K. Joiner be awarded his special damages as are proved at trial plus an appropriate amount as general damages in an amount in excess of Ten Million Dollars ($10,000,000.00.);

(e) Under Count Five, that Plaintiff Robert K. Joiner be awarded his special damages as are proved at trial plus an appropriate amount as general damages in an amount in excess of Ten Million Dollars ($10,000,000.00);

(f) Under Count Six, that Plaintiff Karen P. Joiner be awarded her special damages as are proved at trial plus an appropriate amount as general damages in an amount in excess of Ten Million Dollars ($10,000,000.00);

(g) Under Count Seven, that Plaintiffs be awarded punitive damages in an amount in excess of Twenty Million Dollars ($20,000,000.00.);

(h) That the cost of this action be levied against Defendants; and,

(i) That Plaintiffs be awarded such other and further relief as this Court deems just and proper.

Respectfully Submitted,

BURGE & WETTERMARK

By: /s/ Michael J. Warshauer
MICHAEL J. WARSHAUER
Georgia Bar Number 018720

Suite 3250, One Atlantic Center
1201 W. Peachtree Street, NE
Atlanta, Georgia 30309
(404) 875-2500

[Filed Sep. 4, 1992]

IN THE STATE COURT OF FULTON COUNTY
STATE OF GEORGIA

(Title Omitted in Printing)

ANSWER OF GENERAL ELECTRIC COMPANY TO PLAINTIFFS' COMPLAINT

COMES NOW Defendant GENERAL ELECTRIC COMPANY (hereinafter "General Electric") and answers Plaintiff's Complaint as follows:

First Defense

General Electric answers the numbered paragraphs of the Complaint as follows:

1.

General Electric denies the allegations in this paragraph insofar as they allege that General Electric committed a tortious act in Georgia. For further answer to Paragraph 1 of the Complaint, General Electric admits that it is subject to the jurisdiction and venue of this Court.

2.

General Electric is without knowledge or information sufficient to form a belief as to the truth of the allegations in Paragraph 2 of the Complaint.

3.

General Electric is without knowledge or information sufficient to form a belief as to the truth of the allegations in Paragraph 3 of the Complaint.

4.

General Electric admits that Monsanto manufactured Polychlorinated Biphenyls ("PCB") to be used in electrical transformers manufactured by General Electric. General Electric also manufactured electrical transformers which did not contain PCB's, and therefore, General Electric is without knowledge or information sufficient to form a belief as to the allegation that Robert K. Joiner was exposed to PCB's. Except as thus stated, General Electric is without knowledge or information sufficient to form a belief as to the truth of the remaining allegations in Paragraph 4 of the Complaint.

5.

General Electric admits the allegations in paragraph 5 of the Complaint. General Electric also manufactured electrical transformers which did not contain PCB's, and therefore, General Electric is without knowledge or information sufficient to form a belief as to the allegation that Robert K. Joiner was exposed to PCB's.

6.

General Electric denies the allegations in Paragraph 6 of the Complaint.

7.

General Electric is without knowledge or information sufficient to form a belief as to the truth of the allegations in Paragraph 7 of the Complaint. General Electric also manufactured electrical transformers which did not contain PCB's, and therefore, General Electric is without knowledge or information sufficient to form a belief as to the allegation that Robert K. Joiner was exposed to PCB's.

8.

General Electric is without knowledge or information sufficient to form a belief as to the truth of the allega-

tions in Paragraph 8 of the Complaint. General Electric also manufactured electrical transformers which did not contain PCB's, and therefore, General Electric is without knowledge or information sufficient to form a belief as to the allegation that Robert K. Joiner was exposed to PCB's.

9.

General Electric denies the allegations in Paragraphs 9 of the Complaint.

10.

Answering Paragraph 10 of the Complaint, General Electric incorporates by reference its answers to Paragraphs 1 through 9 of the Complaint as if restated verbatim.

11.

General Electric denies the allegations in Paragraphs 11 of the Complaint.

12.

General Electric denies the allegations in Paragraphs 12 of the Complaint.

13.

Answering Paragraph 13 of the Complaint, General Electric incorporates by reference its answers to Paragraphs 1 through 12 of the Complaint as if restated verbatim.

14.

General Electric denies the allegations in Paragraphs 14 of the Complaint.

15.

General Electric denies the allegations in paragraph 15 of the Complaint.

16.

Answering Paragraph 16 of the Complaint, General Electric incorporates by reference its answers to Para-

graphs 1 through 15 of the Complaint as if restated verbatim.

17.

General Electric denies the allegations in paragraph 17 of the Complaint insofar as they suggest that any representations made by General Electric were false or misleading. General Electric is without knowledge or information sufficient to form a belief as to the truth of the allegations in Paragraph 17 of Complaint regarding Robert K. Joiner.

18.

General Electric denies the allegations in Paragraph 18 insofar as they suggest that any representations made by General Electric were false or misleading. General Electric is without knowledge or information sufficient to form a belief as to the truth of the allegations in Paragraph 18 of Complaint regarding Robert K. Joiner.

19.

General Electric denies the allegations in paragraph 19 of the Complaint insofar as they suggest that any representations made by General Electric were false or misleading. General Electric is without knowledge or information sufficient to form a belief as to the truth of the allegations in Paragraph 19 of Complaint regarding Robert K. Joiner.

20.

General Electric denies the allegations in paragraph 20 of the Complaint.

21.-26.

The allegations of Paragraphs 21 through 26 of the Complaint require no answer by General Electric since it is not a party to Counts Four and Five of the Complaint. However, General Electric specifically denies that any claim for injuries or damages referenced in these paragraphs was caused by General Electric. To the

extent that these allegations require a further answer, General Electric denies the allegations of Paragraphs 21, 22, 23, 24, 25, and 26 of the Complaint.

27.

Answering Paragraph 27 of the Complaint, General Electric incorporates by reference its answers to Paragraphs 1 through 26 of the Complaint as if restated verbatim.

28.

General Electric is without knowledge or information sufficient to form a belief as to the truth of the allegations in Paragraph 28 of Complaint.

29.

General Electric denies the allegations in Paragraph 29 of the Complaint.

30.

General Electric denies the allegations in Paragraph 30 of the Complaint.

31.

Answering Paragraph 31 of the Complaint, General Electric incorporates by reference its answers to Paragraph 1 through 30 of the Complaint as if restated verbatim.

32.

General Electric denies the allegations in Paragraph 32 of the Complaint.

33.

General Electric denies the allegations in Paragraph 33 of the Complaint.

34.

General Electric denies all other allegations in the Complaint to which specific reference has not been made in this answer.

ADDITIONAL DEFENSES

As there are no averments of time and place, as required by O.C.G.A. § 9-11-9(f), General Electric is required to answer the Complaint by raising affirmative defenses which may, or may not, be applicable, depending upon the facts and circumstances. As such, General Electric pleads the defenses which follow; however, General Electric reesrves the right to seasonably amend or supplement its answer upon discovery of facts and circumstances which give rise to additional defenses:

Second Defense

Plaintiff's Complaint, in whole or in part, fails to state a claim against General Electric upon which relief can be granted.

Third Deefnse

Plaintiff Robert K. Joiner's alleged injuries and damages, if as alleged, were not proximately caused by exposure to PCB's.

Fourth Defense

Plaintiff Robert K. Joiner's alleged injuries and damages, if as alleged, were not proximately caused by General Electric.

Fifth Defense

Some or all of Plaintiffs' claims may be barred by the applicable statute of repose; however, there are no allegations of time as required by O.C.G.A. § 9-11-9(f).

Sixth Defense

Some or all of Plaintiffs' claims may be barred by the applicable statute of limitations; however, there are no allegations of time as required by O.C.G.A. § 9-11-9(f).

Seventh Defense

Plaintiffs may be barred from recovery under the doctrine of assumption of the risk.

Eighth Defense

Plaintiffs may be barred from recovery, in whole or in part, due to Robert K. Joiner's negligence, under the doctrines of contributory and comparative negligence.

Ninth Defense

The fraud alleged in Count Three, Paragraphs 16-20, of the Complaint, are not stated with particularity as required by O.C.G.A. § 9(b).

Tenth Defense

Plaintiffs have failed to specifically state items of special damages as required by O.C.G.A. § 9-11-9(g).

Eleventh Defense

If Robert K. Joiner, by the exercise of ordinary care, could have avoided the consequences allegedly caused by General Electric's negligence, then the Plaintiffs are not entitled to recover.

Twelfth Defense

If neither Robert K. Joiner nor General Electric were guilty of negligence, then any injuries or damages would be the result of an accident.

Thirteenth Defense

Plaintiffs' claims for punitive damages are in violation of and barred by the due process clauses of the Georgia and United States Constitutions.

Fourteenth Defense

Plaintiffs' claims for punitive damages are in violation of and barred by the equal protection clauses of the Georgia and United States Constitutions.

WHEREFORE, having fully answered the Complaint, Defendant General Electric demands judgment in its favor as follows:

(1) Dismissing the Complaint with prejudice;

(2) Awarding all costs of litigation to Defendant General Electric; and

(3) Awarding all such other relief to Defendant General Electric as this Court deems proper.

This 4th day of September, 1992.

CHILIVIS & GRINDLER

/s/ Anthony L. Cochran
GARY G. GRINDLER—312650
ANTHONY L. COCHRAN—172425
TIMOTHY J. BUCKLEY, III—092913

Attorneys for General Electric Company

3127 Maple Drive, N.E.
Atlanta, Georgia 30305
(404) 233-4171

IN THE STATE COURT OF FULTON
STATE OF GEORGIA

(Title Omitted in Printing)

ANSWER OF THE DEFENDANT, MONSANTO COMPANY

Comes now MONSANTO COMPANY, (hereinafter "MONSANTO"), Defendant in the above-styled cause, and for Answer to Plaintiffs' Complaint shows the following:

FIRST DEFENSE

In answer to the numbered paragraphs of Plaintiffs' Complaint, Defendant MONSANTO says:

1.

This Defendant is without knowledge or information sufficient to form a belief as to the truth of the allegations contained in Paragraph One (1) of Plaintiffs' Complaint.

2.

This Defendant is without knowledge or information sufficient to form a belief as to the truth of the allegations contained in Paragraph Two (2) of Plaintiffs' Complaint.

3.

With respect to the allegations contained in Paragraph Three (3) of Plaintiffs' Complaint, this Defendant denies that MONSANTO has committed a tortious act in Georgia but admits the remaining allegations contained in Paragraph Three (3).

4.

MONSANTO admits that from time to time it manufactures polychlorinated biphenyls which were sold to General Electric Corporation, among others, as cooling fluid in electrical transformers.

5.

The allegations contained in Paragraph Five (5) of Plaintiffs' Complaint are admitted.

6.

The allegations contained in Paragraph Six (6) of Plaintiffs' Complaint are denied.

7.

The allegations contained in Paragraph Seven (7) of Plaintiffs' Complaint are denied.

8.

The allegations contained in Paragraph Eight (8) of Plaintiffs' Complaint are denied.

9.

The allegations contained in Paragraph Nine (9) of Plaintiffs' Complaint are denied.

COUNT ONE

10.

In response to the allegations contained in Paragraph Ten (10) of COUNT ONE of Plaintiffs' Complaint, Defendant MONSANTO realleges, adopts and incorporates herein its answer to the allegations contained in Paragraphs 1 through 9.

11.

The allegations contained in Paragraph Eleven (11) of COUNT ONE of Plaintiffs' Complaint are denied.

12.

The allegations contained in Paragraph Twelve (12) of COUNT ONE of Plaintiffs' Complaint are denied.

COUNT TWO

13.

In response to the allegations contained in Paragraph Thirteen (13) of COUNT TWO of Plaintiffs' Complaint, Defendant MONSANTO realleges, adopts and incorporates herein its answers to the allegations contained in Paragraphs 1 through 12.

14.

The allegations contained in Paragraph Fourteen (14) of COUNT TWO of Plaintiffs' Complaint require no answer of the Defendant MONSANTO.

15.

The allegations contained in Paragraph Fifteen (15) of COUNT TWO of Plaintiffs' Complaint require no answer of the Defendant MONSANTO.

COUNT THREE

16.

In response to the allegations contained in Paragraph Sixteen (16) of COUNT THREE of Plaintiffs' Complaint, Defendant MONSANTO realleges, adopts and incorporates herein its answers to the allegations contained in Paragraphs 1 through 15

17.

The allegations contained in Paragraph Seventeen (17) of COUNT THREE of Plaintiffs' Complaint require no answer of the Defendant MONSANTO.

18.

The allegations contained in Paragraph Eighteen (18) of COUNT THREE of Plaintiffs' Complaint require no answer of the Defendant MONSANTO.

19.

The allegations contained in Paragraph Nineteen (19) of COUNT THREE of Plaintiffs' Complaint require no answer of the Defendant MONSANTO.

20.

The allegations contained in Paragraph Twenty (20) of COUNT THREE of Plaintiffs' Complaint require no answer of the Defendant MONSANTO.

COUNT FOUR

21.

In response to the allegations contained in Paragraph Twenty-one (21) of COUNT FOUR of Plaintiffs Complaint, Defendant MONSANTO realleges, adopts and incorporates herein its answers to the allegations contained in Paragraphs 1 through 20.

22.

The allegations contained in Paragraph Twenty-two (22) of COUNT FOUR of Plaintiffs' Complaint are denied.

23.

The allegations contained in Paragraph Twenty-three (23) of COUNT FOUR of Plaintiffs' Complaint are denied.

COUNT FIVE

24.

In response to the allegations contained in Paragraph Twenty-four (24) of COUNT FIVE of Plaintiffs' Complaint, Defendant MONSANTO realleges, adopts and incorporates herein its answers to the allegations contained in Paragraphs 1 through 23

25.

The allegations contained in Paragraph Twenty-five (25) of COUNT FIVE of Plaintiffs' Complaint are denied.

26.

The allegations contained in Paragraph Twenty-six (26) of COUNT FIVE of Plaintiffs' Complaint are denied.

COUNT SIX

27.

In response to the allegations contained in Paragraph Twenty-seven (27) of COUNT SIX of Plaintiffs' Complaint, Defendant MONSANTO realleges, adopts and incorporates herein its answers to the allegations contained in Paragraphs 1 through 26.

28.

This Defendant is without knowledge or information sufficient to form a belief as to the truth of the allegations contained in Paragraph Twenty-eight (28) of COUNT SIX of Plaintiffs' Complaint.

29.

The allegations contained in Paragraph Twenty-nine (29) of COUNT SIX of Plaintiffs' Complaint are denied.

30.

The allegations contained in Paragraph Thirty (30) of COUNT SIX of Plaintiffs' Complaint are denied.

COUNT SEVEN

31.

In response to the allegations contained in Paragraph Thirty-one (31) of COUNT SEVEN of Plaintiffs' Complaint, Defendant MONSANTO realleges, adopts and incorporates herein its answers to the allegations contained in Paragraphs 1 through 30.

32.

The allegations contained in Paragraph Thirty-two (32) of COUNT SEVEN of Plaintiffs' Complaint are denied.

33.

The allegations contained in Paragraph Thirty-three (33) of COUNT SEVEN of Plaintiffs' Complaint are denied.

SECOND DEFENSE

The Defendant MONSANTO COMPANY hereby denies any paragraph or any portion of any paragraph not previously referred to herein.

PREAMBLE TO AFFIRMATIVE DEFENSES

The Defendant MONSANTO COMPANY reserves the right to rely upon any of the following defenses to claims asserted by Plaintiffs to the extent that such defenses are supported by information developed through discovery or by evidence at trial:

THIRD DEFENSE

No act or omission on the part of the Defendant MONSANTO either proximately caused or contributed to any injury alleged to have been sustained by Plaintiffs.

FOURTH DEFENSE

Upon information and belief, this Defendant says that Plaintiff ROBERT K. JOINER assumed the risk of injury, if any he sustained, himself and therefore is not entitled to recover of this Defendant.

FIFTH DEFENSE

Defendant MONSANTO says that, even if it was negligent in the manner set out and charged in the Complaint, which alleged negligence is expressly denied, the negligence of Plaintiff ROBERT K. JOINER was equal to or greater than any negligence charged against this Defendant in such Complaint and the consequences of such negligence, if any in fact existed, could have been avoided had ROBERT K. JOINER been in the exercise of ordinary care.

SIXTH DEFENSE

Upon information and belief, Plaintiff ROBERT K. JOINER has failed, in whole or in part, to mitigate and/or avoid any alleged injury purportedly sustained by Plaintiffs.

SEVENTH DEFENSE

Plaintiffs' claims for punitive damages are in violation of and barred by the due process clause of the 1983 Georgia Constitution, Article 1, Section 1, Paragraph 1, and by the Fifth and Fourteenth Amendments to the United States Constitution.

EIGHTH DEFENSE

Plaintiffs' claims for punitive damages are in violation of and barred by the equal protection clause of the 1983 Georgia Constitution, Article 1, Section 1, Paragraph 2, and by the Fourteenth Amendment to the United States Constitution.

NINTH DEFENSE

Upon information and belief, if Plaintiff ROBERT K. JOINER was exposed to polychlorinated biphenyls

("PCBs") alleged to have been manufactured by MONSANTO and if Plaintiff ROBERT K. JOINER incurred any injury resulting therefrom, all of which is expressly denied, Plaintiff ROBERT K. JOINER's injury would not have occurred but for the fact that the "PCBs" were materially changed or altered by Plaintiff ROBERT K. JOINER or others, whether or not such others are parties to this action, and such material change or alteration was not made in accordance with the instructions of MONSANTO, was not made with the consent of MONSANTO or was not the result of conduct that reasonably should have been anticipated by MONSANTO, and therefore Plaintiff ROBERT K. JOINER's claim is barred.

TENTH DEFENSE

Upon information and belief, if Plaintiff ROBERT K. JOINER sustained any injury or damage as alleged in the Complaint, which is denied, such injury was due to, caused by or contributed to, in whole or in part, to the negligence or carelessness of other parties over whom Monsanto had no control and for whose conduct MONSANTO is not responsible. Such negligence of other parties was the sole proximate cause of, or an intervening, superseding, or insulating cause of the Plaintiffs' alleged damages.

ELEVENTH DEFENSE

Upon information and belief, it is alleged that the Plaintiff ROBERT K. JOINER's employer at the time of the alleged exposure to "PCBs" was negligent in that the employer failed properly and safely to equip the Plaintiff ROBERT K. JOINER with the necessary protection; that the employer provided the orders and directions under which the Plaintiff ROBERT K. JOINER worked with or in the area of "PCBs" (if he did so); that the employer allowed "PCBs" to be used by the Plaintiff ROBERT K. JOINER and others in a manner so as to create a condition of danger for the Plaintiff ROBERT K. JOINER; that

although the employer knew or should have known in the exercise of ordinary care of the general requirements of the handling of "PCBs", the employer nevertheless failed to relate all such information to the Plaintiff ROBERT K. JOINER; that the employer failed to provide a safe place for the Plaintiff ROBERT K. JOINER to work; that the employer failed to provide suitable training and education for its employees, including the Plaintiff ROBERT K. JOINER; that the employer failed to require the Plaintiff ROBERT K. JOINER and others to keep the premises and their persons clean and in a normal state of good housekeeping and cleanliness. If the Plaintiff ROBERT K. JOINER sustained any injury or damage as set forth in the Complaint, which is denied, the same was directly and proximately caused by the foregoing negligence of the Plaintiff ROBERT K. JOINER's employer and such negligence was the sole proximate cause of or an intervening, superseding or insulating cause of the Plaintiff ROBERT K. JOINER's injury and damage, if any.

TWELFTH DEFENSE

Any purchaser or user who purchased or used "PCBs" alleged to have been manufactured by MONSANTO, as the developers and manufacturers of the final product to which the Plaintiff ROBERT K. JOINER was allegedly exposed, were sophisticated and knowledgeable users of "PCBs" and were in a far better position to instruct or warn Plaintiff ROBERT K. JOINER and, if any such instruction or warning was legally required, which is denied, their failure to instruct or warn the Plaintiff ROBERT K. JOINER was an intervening, superseding or insulating proximate cause of Plaintiff ROBERT K. JOINER's alleged injuries.

THIRTEENTH DEFENSE

If Plaintiff ROBERT K. JOINER was exposed to any "PCBs" alleged to have been manufactured by MONSANTO and if Plaintiff ROBERT K. JOINER incurred

any injury resulting therefrom, all of which is expressly denied, Plaintiff ROBERT K. JOINER's claim is barred by the applicable statute of limitations.

WHEREFORE, the Defendant MONSANTO COMPANY respectfully demands that judgment be rendered in its favor.

FREEMAN & HAWKINS

/s/ Joe C. Freeman, Jr.
JOE C. FREEMAN, JR.
Ga. State Bar No. 275700

/s/ Joanne Beauvoir Brown
JOANNE BEAUVOIR BROWN
Ga. State Bar No. 045730
Attorneys for Defendant
Monsanto Company

2800 First Atlanta Tower
Atlanta, Georgia 30383
(404) 522-0856

IN THE STATE COURT OF FULTON COUNTY
STATE OF GEORGIA

(Title Omitted in Printing)

ANSWER OF DEFENDANT WESTINGHOUSE ELECTRIC CORPORATION

COMES NOW defendant Westinghouse Electric Corporation and hereby raises its defenses and answers plaintiffs' complaint as follows:

FIRST DEFENSE

Plaintiffs' complaint fails to state a claim upon which relief may be granted.

SECOND DEFENSE

Plaintiffs' complaint is barred by the statute of limitations.

THIRD DEFENSE

Plaintiffs' complaint is barred by the statute of repose.

FOURTH DEFENSE

Plaintiffs are barred from any recovery under the doctrine of assumption of risk.

FIFTH DEFENSE

Plaintiffs are barred from any recovery in whole or in part by the contributory negligence or comparative fault of plaintiffs.

SIXTH DEFENSE

The allegations of fraud set forth in count three, paragraphs 16-20, of the plaintiff's complaint, are not stated with the requisite particularity.

SEVENTH DEFENSE

The plaintiffs have failed to specifically state items of special damages.

EIGHTH DEFENSE

Having raised its affirmative defenses and without waiving said defenses, defendant Westinghouse responds to each and every paragraph of the plaintiffs' complaint as follows:

1.

Defendant Westinghouse is without sufficient knowledge or information upon which to form a belief as to the truth or accuracy of the allegations contained in paragraph 1 of the complaint.

2.

Defendant Westinghouse admits that it is a Pennsylvania corporation, that it maintains a registered agent in Fulton County, Georgia, and that it is subject to the jurisdiction and venue of this Court. Defendant Westinghouse denies that it committed a tortious act in Georgia as alleged in plaintiffs' complaint and denies all remaining allegations of paragraph 2 of the plaintiffs' complaint.

3.

Defendant Westinghouse is without sufficient knowledge or information upon which to form a belief as to the truth or accuracy of the allegations contained in paragraph 3 of the complaint.

4.

Defendant Westinghouse admits that it purchased from Monsanto Company quantities of polychlorinated biphenyls for use in certain electrical transformers it manufactured. Defendant Westinghouse denies the remaining allegations of paragraph 4 of plaintiffs' complaint.

5.

Defendant Westinghouse admits that it was a manufacturer of electrical transformers which contained polychlorinated biphenyls and that some of these transformers contained polychlorinated biphenyls purchased from Monsanto Company. Defendant Westinghouse is without sufficient knowledge or information upon which to form a belief as to the truth or accuracy of the plaintiffs' allegations concerning General Electric Company.

6.

Defendant Westinghouse denies the allegations contained in paragraph 6 of the complaint.

7.

Defendant Westinghouse denies the allegations contained in paragraph 7 of the complaint.

8.

Defendant Westinghouse denies the allegations contained in paragraph 8 of the complaint.

9.

Defendant Westinghouse denies the allegations contained in paragraph 9 of the complaint.

10.

In response to paragraph 10 of plaintiffs' complaint, defendant Westinghouse realleges and incorporates herein

its responses to paragraphs 1 through 9 of the complaint with the same force and effect as if they had been restated herein.

11.

Defendant Westinghouse denies the allegations contained in paragraph 11 of the complaint.

12.

Defendant Westinghouse denies the allegations contained in paragraph 12 of the complaint.

13.

In response to paragraph 13 of plaintiffs' complaint, defendant Westinghouse realleges and incorporates herein its responses to paragraphs 1 through 12 of the complaint with the same force and effect as if they had been restated herein.

14.

Defendant Westinghouse denies the allegations contained in paragraph 14 of the complaint.

15.

Defendant Westinghouse denies the allegations contained in paragraph 15 of the complaint.

16.

In response to paragraph 16 of plaintiffs' complaint, defendant Westinghouse realleges and incorporates herein its responses to paragraphs 1 through 15 of the complaint with the same force and effect as if they had been restated herein.

17.

Defendant Westinghouse denies the allegations contained in paragraph 17 of the complaint.

18.

Defendant Westinghouse denies the allegations contained in paragraph 18 of the complaint.

19.

Defendant Westinghouse denies the allegations contained in paragraph 19 of the complaint.

20.

Defendant Westinghouse denies the allegations concontained in paragraph 20 of the complaint.

21.

In response to paragraph 21 of plaintiffs' complaint, defendant Westinghouse realleges and incorporates herein its responses to paragraphs 1 through 20 of the complaint with the same force and effect as if they had been restated herein.

22.

Defendant Westinghouse denies the allegations contained in paragraph 22 of the complaint.

23.

Defendant Westinghouse denies the allegations contained in paragraph 23 of the complaint.

24.

In response to paragraph 24 of plaintiffs' complaint, defendant Westinghouse realleges and incorporates herein its responses to paragraphs 1 through 23 of the complaint with the same force and effect as if they had been restated herein.

25.

Defendant Westinghouse denies the allegations contained in paragraph 25 of the complaint.

26.

Defendant Westinghouse denies the allegations contained in paragraph 26 of the complaint.

27.

In response to paragraph 27 of plaintiffs' complaint, defendant Westinghouse realleges and incorporates herein its responses to paragraphs 1 through 26 of the complaint with the same force and effect as if they had been restated herein.

28.

Defendant Westinghouse is without sufficient knowledge or information upon which to form a belief as to the truth or accuracy of the allegations contained in paragraph 28 of the complaint.

29.

Defendant Westinghouse denies the allegations contained in paragraph 29 of the complaint.

30.

Defendant Westinghouse denies the allegations contained in paragraph 30 of the complaint.

31.

Defendant Westinghouse is without sufficient knowledge or information upon which to form a belief as to the truth or accuracy of the allegations contained in paragraph 31 of the complaint.

32.

Defendant Westinghouse denies the allegations contained in paragraph 32 of the complaint.

33.

Defendant Westinghouse denies the allegations contained in paragraph 33 of the complaint.

WHEREFORE, having asserted its affirmative defenses and having answered plaintiffs' complaint, defendant Westinghouse demands judgment that the plaintiffs' complaint be dismissed with all costs cast against plaintiffs.

This the 4th day of September, 1992.

/s/ David H. Flint
DAVID H. FLINT
Georgia Bar No. 264600
ALEX J. SIMMONS, JR.
Georgia Bar No. 646990

Attorneys for defendant
Westinghouse Electric Corp.

SCHREEDER, WHEELER & FLINT
1600 Candler Building
127 Peachtree Street, N.E.
Atlanta, Georgia 30303-1845
(404) 681-3450

[Filed Sep. 4, 1992]

IN THE UNITED STATES DISTRICT COURT
FOR THE NORTHERN DISTRICT OF GEORGIA
ATLANTA DIVISION

Civil Action File No. 1 92.CV.2137

ROBERT K. JOINER, and
KAREN P. JOINER,
Plaintiffs

vs.

GENERAL ELECTRIC COMPANY,
A New York Corporation;
WESTINGHOUSE ELECTRIC CORPORATION,
a Pennsylvania corporation;
and MONSANTO COMPANY,
a Delaware corporation,
Defendants

PETITION FOR REMOVAL

The petition of GENERAL ELECTRIC COMPANY, WESTINGHOUSE ELECTRIC CORPORATION and MONSANTO COMPANY for the removal of the action herein referred to from the State Court of Fulton County, Georgia, to the United States District Court for the Northern District of Georgia, Atlanta Division, respectfully shows to the Court the following:

1.

That Petitioners herein, GENERAL ELECTRIC COMPANY, WESTINGHOUSE ELECTRIC CORPORATION and MONSANTO COMPANY, are Defendants in a civil action brought in the State Court of Fulton County,

Georgia, entitled *Robert K. Joiner, and Karen P. Joiner vs. General Electric Company, a New York Corporation, Westinghouse Electric Corporation, a Pennsylvania Corporation, and Monsanto Company, a Delaware Corporation,* Civil Action No. 92 VS 61273A. Attached hereto and made a part hereof are:

1. Exhibit "A"—A true copy of the original Summons & Complaint filed by Plaintiffs in the State Court of Fulton County, Georgia;

2. Exhibit "B"—A true copy of the Answer filed in the State Court of Fulton County, Georgia by Defendant WESTINGHOUSE ELECTRIC CORPORATION;

3. Exhibit "C"—Notice of Removal filed in the State Court of Fulton County, State of Georgia, by Petitioners.

4. Exhibit "D"—A true copy of the Answer filed in the State Court of Fulton County, Georgia by Defendant GENERAL ELECTRIC COMPANY;

5. Exhibit "E"—A true copy of the Answer filed in the State Court of Fulton County, Georgia by Defendant MONSANTO COMPANY;

The attachments hereto, Exhibits "A" "B" "C" "D" and "E" constitute all of the pleadings which have now been filed in the subject case in the said State Court of Fulton County.

2.

That said action was commenced by service upon Defendant WESTINGHOUSE ELECTRIC CORPORATION, through Prentice Hall Corporation System on August 7, 1992 and upon Defendants MONSANTO COMPANY and GENERAL ELECTRIC COMPANY, through C. T. Corporation System on August 10, 1992. This petition is filed within thirty (30) days from the date of service upon these Defendants in this case, pursuant to 28 USC § 1446.

3.

That the controversy between the Plaintiffs and your Petitioners is a controversy between residents of the State of Georgia and three corporations, residents of the States of New York, Pennsylvania and Missouri respectively.

4.

That upon information and belief, Plaintiffs, ROBERT K. JOINER and KAREN P. JOINER, were, at the time of the commencement of this action, and still are citizens and residents of the state of Georgia;

5.

Defendant GENERAL ELECTRIC COMPANY, a New York corporation was, at the time of the commencement of this action, and still is a corporation with its principal place of doing business located at Fairfield, Connecticut.

6.

Defendant WESTINGHOUSE ELECTRIC CORPORATION, a Pennsylvania corporation was, at the time of the commencement of this action, and still is, a corporation with its principal place of doing business located at Pittsburgh, Pennsylvania.

7.

Defendant MONSANTO COMPANY, a Delaware corporation, was at the time of the commencement of this action, and still is, a corporation with its principal place of doing business located at St. Louis, Missouri.

8.

That the matter in controversy exceeds the value of Fifty Thousand Dollars ($50,000.00) exclusive of interest and costs, and is a civil action brought in a State Court of the State of Georgia, of which the United States District Courts have original jurisdiction because of diver-

sity of citizenship and the amount in controversy pursuant to 28 USC § 1332.

6.

That the pending action is one which Defendants, GENERAL ELECTRIC CORPORATION, WESTINGHOUSE ELECTRIC CORPORATION and MONSANTO COMPANY, are entitled to remove to this Court pursuant to 28 USC § 1441.

WHEREFORE, Petitioners pray that this Petition for Removal be filed and that said action be removed to and proceed in this Court and that no further proceedings be had in the said case in the State Court of Fulton County, Georgia.

[Counsel omitted in printing.]

[Filed Oct. 27, 1992]

IN THE UNITED STATES DISTRICT COURT
FOR THE NORTHERN DISTRICT OF GEORGIA
ATLANTA DIVISION

(Title Omitted in Printing)

PLAINTIFFS' ANSWERS TO MANDATORY INTERROGATORIES

* * * *

EXPERT WITNESSES

* * * *

12) Dr. Larry W. Robertson, Associate Professor of Toxicology, Graduate Center for Toxicology, University of Kentucky, 204 Funkhouser Boulevard, Lexington, Kentucky 40506-0054. Dr. Robertson is expected to testify as to medical causation, specifically, that Plaintiff Robert K. Joiner's exposure to PCBs is a direct and proximate cause of his lung cancer. Dr. Robertson will also attest to his analysis of Triangle Laboratories' data and toxicology reports concerning the presence of PCBs in Plaintiff Robert K. Joiner's fat tissue. Dr. Robertson's testimony may be introduced by deposition.

As discovery is ongoing, Plaintiffs reserve the right to supplement their Witness List.

* * * *

[Filed in Chambers 7/9/93]

IN THE UNITED STATES DISTRICT COURT
FOR THE NORTHERN DISTRICT OF GEORGIA
ATLANTA DIVISION

(Title Omitted in Printing)

JOINT REQUEST FOR CONTINUANCE AND FOR SCHEDULING ORDER

All of the parties having consented, they collectively request that this Court extend the time for discovery and enter a scheduling order for the following reasons:

This is a tort action in which Robert and Karen Joiner contend that his lung cancer was caused by alleged exposure to certain chemicals. The Defendants deny liability for a number of reasons; however, the central issues in this case relate to causation of Robert K. Joiner's illness. As the Court would expect, the Plaintiffs and Defendants vigorously disagree over the issue of causation. While there are innumerable issues which could get litigated and discovered in this case (at great expense), there is one potentially dispositive issue, *i.e.*, causation of Robert K. Joiner's illness.

Causation must be established by expert testimony, *i.e.*, toxicologists, epidemiologists, analytical chemists and physicians in various specialties. It is appropriate for the Court to consider the proposed scheduling order in the interests of judicial economy. Additionally, the parties would hope to avoid unnecessary fees and expenses by first having this Court consider whether there is an issue of fact on the potentially dispositive issue of causation. A scheduling order addressing the issue of causation can be addressed first and resolved before the parties engage in expensive discovery and litigation on numerous other issues.

* * * *

The following facts support the requested extension and scheduling order:

1.

This case was removed to federal court on September 4, 1992.

2.

On October 26, 1992, the plaintiffs served their answers to the Court's mandatory interrogatories.

3.

In the plaintiffs' mandatory interrogatory answers, they identified one expert witness on the issue of medical causation, Dr. Larry W. Robertson, a toxicologist.

4.

On December 11, 1992, the defendants served their answers to the Court's mandatory interrogatories. At that time, the defendants identified several expert witnesses whose testimony will touch on the issue of medical causation: Dr. Philip Cole, Chairman of Department Epidemiology in the School of Public Health and Associate Director for Epidemiology of the Comprehensive Cancer Center at the University of Alabama at Birmingham; Dr. William J. Waddell, Chairman of the Department of Pharmacology and Toxicology at the University of Louisville School of Medicine; Dr. George Frame, a Research Scientist and Analytical Chemist with General Electric.

5.

On January 8, 1993, in interrogatory answers, General Electric identified Dr. Stephen B. Hamilton, Jr., Manager Science and Technology with General Electric as an additional expert with knowledge concerning research and testing conducted or sponsored by General Electric relating to PCB exposure among humans.

6.

On February 1, 1993, the parties consented to an extention of the discovery period through June 4 as additional time was necessary for the completion of discovery as a result of the Thanksgiving, Christmas and New Year's holidays.

7.

On April 1, 1993, the plaintiffs served their first supplemental response to this Court's mandatory interrogatories identifying Dr. Arnold J. Schecter as an additional expert witness on the issue of causation.

8.

On June 1, 1993, an additional extension of time was jointly requested by the parties to extend the discovery period due to complications in scheduling and other circumstances beyond the parties' control. The discovery period was extended through August 3, 1993.

9.

On June 9, 1993, the depositions of Robert and Karen Joiner were taken in Thomasville, Georgia.

10.

On June 23, 1993, the defendants supplemented their discovery responses identifying Dr. William C. Bailey, a Professor of Medicine at the University of Alabama School of Medicine in Birmingham, Alabama as an additional expert witness on the issue of causation.

11.

On June 29, 1993, the plaintiffs filed a motion to extend discovery through October 31, 1993.

12.

On June 30, 1993 the plaintiffs served their second supplemental response to this Court's mandatory interrog-

atories identifying two additional expert witnesses on the issue of causation: Dr. Daniel T. Teitelbaum, a Clinical Toxicologist, and Dr. Arthur L. Frank, a professor in the Department of Preventive Medicine and Environmental Health at the University of Kentucky College of Medicine.

13.

Depositions of the following witnesses are currently scheduled:

a. Dr. Robertson's deposition is currently scheduled for July 13 in Lexington, Kentucky;

b. Dr. Schecter's deposition in Binghamton, New York was originally scheduled for July 16 but was recheduled for July 14 at Dr. Schecter's request;

c. The deposition of Dr. Thomas O. Rouse of General Electric is currently scheduled for Tuesday, July 20, 1993 in Albany, New York;

d. The deposition of Mr. William Papageorge of Monsanto is scheduled for July 22, 1993 in St. Louis, Missouri;

e. The depositions of Drs. Frame and John F. Brown, Jr. of General Electric are scheduled for July 26, 1993 in Albany, New York;

f. The deposition of Dr. Stephen B. Hamilton, Jr., of General Electric, is scheduled for July 28, 1993 in Trumbull, Connecticut;

g. The deposition of Dr. Bailey is tentatively scheduled for August 19, 1993 in Birmingham, Alabama.

Some of these depositions may not bear on the issue of causation and may be unnecessary if the requested scheduling order is granted.

14.

The parties' desire to reschedule Dr. Robertson's deposition due to the difficult travel arrangements necessary to

travel from Lexington, Kentucky on the evening of July 13 (following Dr. Robertson's deposition of July 13) to Binghamton, New York for Dr. Schecter's deposition on July 14. While it is logistically possible (there is one available night flight, assuming good weather), it would make more sense to extend the discovery deadline beyond August 3 so that Dr. Robertson's deposition can be conveniently rescheduled.

15.

The depositions for the following additional witnesses will need to be scheduled on the issue of causation:

a. The deposition of Dr. Teitelbaum from Denver, Colorado will need to be taken;

b. The deposition of Dr. Frank from Lexington, Kentucky will need to be taken;

c. The deposition of Dr. Cole from Birmingham, Alabama will need to be taken;

d. The deposition of Dr. Waddell from Louisville, Kentucky will need to be taken.

16.

Counsel for Plaintiffs and General Electric both have family vacations scheduled for the first two weeks of August, 1993.

CONCLUSION:

For the foregoing reasons, the parties collectively request that this Court enter a Scheduling Order requiring that all parties identify any and all expert witnesses on the issue of causation of Robert Joiner's illness on or before September 1, 1993, and that the depositions of all expert witnesses on the issue of causation be scheduled and taken on or before November 1, 1993. The parties further collectively request that a deadline of Wednesday, December

1, 1993 be set for the filing of any motions in limine or for summary judgment on issues relating to causation of Robert K. Joiner's illness. Until further order of this Court, discovery on issues unrelated to causation shall be stayed pending a ruling on any motion(s) for summary judgment. However, to prevent duplicate depositions and unnecessary expense, the parties may inquire into all areas about which a witness has knowledge. If an issue of fact is found to exist by this Court with respect to causation, the parties will then be allowed a reasonable discovery period for other issues including, but not limited to, negligence, strict liability, and damages.

Respectfully submitted this 9th day of July, 1993.

Counsel for General Electric

/s/ Anthony L. Cochran
ANTHONY L. COCHRAN
Georgia Bar No. 172425

CHILIVIS & GRINDLER
3127 Maple Drive, N.E.
Atlanta, Georgia 30305
(404) 233-4171

Counsel for Plaintiffs

/s/ Michael J. Warshauer
by /s/ Anthony L. Cochran
w/express permission
MICHAEL J. WARSHAUER
Georgia Bar No. 018720

BURGE & WETTERMARK
Suite 3250, One Atlantic Center
1201 W. Peachtree Street, N.E.
Atlanta, Georgia 30309
(404) 875-2500

Counsel for Westinghouse

/s/ Alexander J. Simmons, Jr.
by /s/ ALC
w/express permission
ALEXANDER J. SIMMONS, JR.
Georgia Bar. No. 646990

SCHREEDER, WHEELER & FLINT
1600 Candler Building
127 Peachtree Street, N.E.
Atlanta, Georgia 30303-1845
(404) 681-3450

Counsel for Monsanto

/s/ Joanne B. Brown
by /s/ ALC
w/express permission
JOANNE B. BROWN
Georgia Bar No. 045730

FREEMAN & HAWKINS
4000 One Peachtree
303 Peachtree Street, N.E.
Atlanta, Georgia 30308-3243
(404) 614-7400

[Filed Jul. 12, 1993]

IN THE UNITED STATES DISTRICT COURT
FOR THE NORTHERN DISTRICT OF GEORGIA
ATLANTA DIVISION

(Title Omitted in Printing)

ORDER

The parties having consented,

IT IS HEREBY ORDERED that all parties shall identify any and all expert witnesses on the issue of causation of Robert K. Joiner's illness on or before September 1, 1993, and that the depositions of all expert witnesses on the issue of causation shall be scheduled and taken on or before November 1, 1993. It is hereby further ordered that on or before Wednesday, December 1, 1993, all motion(s) in limine or for summary judgment on issues relating to causation of Robert K. Joiner's illness shall be filed. Until further order of this Court, discovery on issues unrelated to causation shall be stayed pending a ruling on any motions in limine or for summary judgment. However, to prevent duplicate depositions and unnecessary expense, the parties may inquire into all areas about which a witness has knowledge. If an issue of fact is found to exist by this Court with respect to causation, the parties will then be allowed a reasonable period for discovery of other issues including, but not limited to, negligence, strict liability, damages, and the defendants' denial of liability for plaintiffs' injuries.

SO ORDERED, this 13 day of July, 1993.

/s/ Orinda D. Evans
ORINDA D. EVANS
Judge
United States District Court
for the Northern District of
Georgia

IN THE UNITED STATES DISTRICT COURT
FOR THE NORTHERN DISTRICT OF GEORGIA
ATLANTA DIVISION

(Title Omitted in Printing)

FIRST AMENDED COMPLAINT

NOW COME Plaintiffs, in compliance with this Court's Order dated August 4, 1993, and file this First Amended Complaint against Defendants as follows:

1.

Defendant, General Electric Company, a New York corporation, ("GE") maintains its registered agent in Fulton County, Georgia, committed a tortuous act in Georgia, and is subject to the jurisdiction of the courts of this state, venue in this Court is proper, and it may be served with Summons and Complaint as allowed by law.

2.

Defendant, Westinghouse Electric Corporation, a Pennsylvania corporation ("Westinghouse") maintains its registered agent in Fulton County, Georgia, committed a tortuous act in Georgia, and is subject to the jurisdiction of the courts of this state, venue in this Court is proper, and it may be served with Summons and Complaint as allowed by law.

3.

Defendant, Monsanto Company, a Delaware corporation, ("Monsanto") maintains its registered agent in Fulton County, Georgia, committed a tortuous act in Georgia, and is subject to the jurisdiction of the courts of this

state, venue in this Court is proper, and it may be served with Summons and Complaint as allowed by law.

4.

Defendant Monsanto manufactured Polychlorinated Biphenyls ("PCB") to be used in the manufacturing, testing and as a cooling fluid in electrical transformers manufactured by Defendants GE and Westinghouse.

5.

Defendants GE and Westinghouse manufactured and sold as new products electrical transformers containing PCB fluid manufactured by Defendant Monsanto.

6.

At the time of the manufacturing of these transformers with PCB fluid used in the manufacturing, testing and ultimate product, all Defendants were aware that PCBs were unreasonably dangerous and likely to cause injury and death to human beings who were exposed to the PCB fluids, including PCB residues in mineral oil fluids, during service and use of the transformer products.

7.

Plaintiff Robert K. Joiner was an ultimate, foreseeable, intended user and service of the transformer products manufactured by Defendants GE and Westinghouse which contained PCB fluids, and PCB contamination, manufactured by Defendant Monsanto.

8.

Throughout the course of his use of the transformers in his capacity as a repairman, Plaintiff Robert K. Joiner was caused to be exposed to PCB, was caused to be exposed to PCB fumes, and was caused to inhale PCB con-

taminated fluids, mists, liquids, and steams and he was also caused to absorb PCB through contact with his skin.

9.

As a result of Plaintiff Robert K. Joiner's exposure to PCBs, he contracted terminal lung cancer.

COUNT ONE
(STRICT LIABILITY AGAINST DEFENDANTS GE AND WESTINGHOUSE)

10.

Plaintiffs repeat, reallege, and incorporate by reference, the allegations of paragraphs 1 through 9 as if repeated herein as paragraph 10.

11.

Defendants put an unreasonably dangerous and defective product, the PCB contaminated electrical transformers, into the stream of commerce. The defect was the proximate cause of a serious bodily injury sustained by Plaintiff Robert K. Joiner. As a result, Defendants are strictly liable to Plaintiff.

12.

As a direct and proximate result of Defendants' acts, Plaintiff Robert K. Joiner was injured and suffered damages including permanent physical disfigurement, permanent physical disability, past pain and suffering, future pain and suffering, past medical expenses and lost wages, and future medical expenses and lost wages, all entitling him to an award of damages in his favor and against Defendants, and each of them, jointly and severally, in an amount in excess of Ten Million Dollars ($10,000,000.00).

COUNT TWO
(NEGLIGENCE AGAINST DEFENDANTS GE AND WESTINGHOUSE)

13.

Plaintiffs repeat, reallege, and incorporate by reference, the allegations of paragraphs 1 through 12 as if repeated herein as paragraph 13.

14.

Defendants GE and Westinghouse negligently manufactured and distributed the electrical transformers in that: (i) the electrical transformers were sold in a condition which allowed it to be contaminated with PCBs and cause serious injury in expected use; (ii) the electrical transformers were defective in that they were manufactured and sold with PCB contamination which was likely to cause injury in the use for which they were intended; and (iii) the electrical transformers were sold without adequate warnings and instructions with respect to the known and unknown dangers of PCBs contained within them. This negligence was the proximate cause of a serious bodily injury sustained by Plaintiff.

15.

As a direct and proximate result of Defendant GE's and Westinghouse's negligence, Plaintiff was injured and has suffered damages including permanent physical disfigurement, permanent physical disability, past pain and suffering, future pain and suffering, past medical expenses and lost wages, and future medical expenses and lost wages, all entitling him tc an award of damages in his favor and against Defendants, and each of them, jointly and severally, in an amount in excess of Ten Million Dollars ($10,000,000.00).

COUNT THREE
(FRAUD AGAINST DEFENDANTS GE AND WESTINGHOUSE)

16.

Plaintiffs repeat, reallege, and incorporate by reference, the allegations of paragraphs 1 through 15 as if repeated herein as paragraph 16.

17.

Defendants GE and Westinghouse represented to the buying public and those who serviced their electrical transformer products, including Plaintiff Robert K. Joiner, that the electrical transformer products were free from PCBs and were safe in regular use and servicing.

18.

Defendants GE and Westinghouse intended for the buying public and those who serviced their electrical transformer products, including Plaintiff Robert K. Joiner, to rely on their representations that the electrical transformer products were free from PCB and were safe in regular use and servicing.

19.

The buying public and those who serviced their electrical transformer products, including and particularly Plaintiff Robert K. Joiner, reasonably relied on the representations of Defendants GE and Westinghouse that the electrical transformer products were free from PCBs and were safe in regular use and servicing and as a result Plaintiff Robert K Joiner took no precautions to protect himself when handling and servicing the electrical transformer products.

20.

As a direct and proximate result of the justifiable reliance on Defendant GE's and Westinghouse's fraudulent misrepresentations, Plaintiff Robert K. Joiner was exposed

to PCBs and was caused to contract terminal lung cancer and was otherwise injured and has suffered damages including permanent physical disfigurement, permanent physical disability, past pain and suffering, future pain and suffering, past medical expenses and lost wages, and future medical expenses and lost wages, all entitling him to an award of damages in his favor and against Defendants, and each of them, jointly and severally, in an amount in excess of Ten Million Dollars ($10,000,000.00).

COUNT FOUR
(STRICT LIABILITY AGAINST DEFENDANT MONSANTO)

21.

Plaintiffs repeat, reallege, and incorporate by reference, the allegations of paragraphs 1 through 20 as if repeated herein as paragraph 21.

22.

Defendant Monsanto put an unreasonably dangerous and defective product, the PCB fluids, into the stream of commerce. The defect was the proximate cause of a serious bodily injury sustained by Plaintiff Robert K. Joiner. As a result, Defendants are strictly liable to Plaintiff.

23.

As a direct and proximate result of Defendant Monsanto's acts, Plaintiff Robert K. Joiner was injured and suffered damages including permanent physical disfigurement, permanent physical disability, past pain and suffering, future pain and suffering, past medical expenses and lost wages, and future medical expenses and lost wages, all entitling him to an award of damages in his favor and against Defendants, and each of them, jointly and severally, in an amount in excess of Ten Million Dollars ($10,000,000.00).

COUNT FIVE
(NEGLIGENCE AGAINST DEFENDANT MONSANTO)

24.

Plaintiffs repeat, reallege, and incorporate by reference, the allegations of paragraph 1 through 23 as if repeated herein as paragraph 24.

25.

Defendant Monsanto negligently manufactured and distributed the PCB fluid in that: (i) the PCB fluid was unreasonably dangerous and known to be likely to cause serious injury in expected use; (ii) the PCB fluid was sold without adequate warnings and instructions with respect to the known and unknown dangers of PCBs. This negligence was the proximate cause of a serious bodily injury sustained by Plaintiff.

26.

As a direct and proximate result of Defendant Monsanto's negligence, Plaintiff was injured and has suffered damages including permanent physical disfigurement, permanent physical disability, past pain and suffering, future pain and suffering, past medical expenses and lost wages, and future medical expenses and lost wages, all entitling him to an award of damages in his favor and against Defendants, and each of them, jointly and severally, in an amount in excess of Ten Million Dollars ($10,000,000.00).

COUNT SIX
(LOSS OF CONSORTIUM AGAINST ALL DEFENDANTS)

27.

Plaintiffs repeat, reallege, and incorporate by reference, the allegations of paragraphs 1 through 26 as if repeated herein as paragraph 27.

28.

At all times relevant to this action, Plaintiff Karen P. Joiner has been the wife of Plaintiff Robert K. Joiner.

29.

As a direct and proximate result of the wrongful conduct of Defendants, and each of them, Plaintiff Robert K. Joiner suffered severe and permanent bodily injuries including serious and permanent physical and emotional injury and disability, substantial and continuing pain, suffering, and discomfort, and lost wages and medical expenses, both past and future. As a result of these injuries to her husband, Plaintiff Karen P. Joiner has lost the domestic and conjugal services previously provided to her by her husband and has been deprived of her right to consortium.

30.

As a direct and proximate result of Defendants' wrongful conduct, Plaintiff Karen P. Joiner is entitled to an award of general damages for her loss of consortium in an amount to be determined by a jury of her peers in an amount in excess of Ten Million Dollars ($10,000,000.00) against each of them, jointly and severally.

COUNT SEVEN
(BATTERY AGAINST ALL DEFENDANTS)

31.

Plaintiffs repeat, reallege, and incorporate by reference, the allegations of paragraphs 1 through 30 as if repeated herein as paragraph 31.

32.

Defendants caused a harmful product to contact Plaintiff Robert K. Joiner which contact was not necessary, was not privileged, and constituted a harmful or offensive contact, consisting of a battery to Plaintiff's person in wanton disregard of Plaintiff's rights.

33.

As a direct and proximate result of Defendants' battery of Plaintiff's person, Plaintiff Robert K. Joiner Plaintiff was injured and has suffered damages including permanent physical disfigurement, permanent physical disabiilty, past pain and suffering, future pain and suffering, past medical expenses and lost wages, and future medical expenses and lost wages, all entitling him to an award of damages in his favor and against Defendants, and each of them, jointly and severally, in an amount in excess of Ten Million Dollars ($10,000,000.00).

COUNT EIGHT
(PUNITIVE DAMAGES AGAINST ALL DEFENDANTS)

34.

Plaintiffs repeat, reallege, and incorporate by reference, the allegations of paragraphs 1 through 33 as if repeated herein as paragraph 34.

35.

Defendants' conduct in defrauding Plaintiff is intentional, in bad faith, and in wanton disregard of Plaintiff's rights. Defendants' conduct in distributing and selling defective and dangerous products which Defendants knew were likely to cause bodily injury was willful, wanton, and in callous disregard for human health and safety.

36.

As a direct and proximate result of Defendants' wrongful conduct, Plaintiffs are entitled to judgment against Defendants, and each of them, jointly and severally, for exemplary damages to deter each of them from similar conduct in the future and to punish them for their wrongful acts in an amount to be determined by the enlightened conscience of the finder of fact in an amount in excess of Twenty Million Dollars ($20,000,000.00).

WHEREFORE, Plaintiffs pray that summons issue, that Defendants be served and made to appear and answer, that a JURY TRIAL be held, and that they be awarded judgment in their favor and against the Defendants, jointly and severally, as follows:

(a) Under Count One, that Plaintiff Robert K. Joiner be awarded his special damages as are proved at trial plus an appropriate amount as general damages in an amount in excess of Ten Million Dollars ($10,000,000.00);

(b) Under Count Two, that Plaintiff Robert K. Joiner be awarded his special damages as are proved at trial plus an appropriate amount as general damages in an amount in excess of Ten Million Dollars ($10,000,000.00);

(c) Under Count Three, that Plaintiff Robert K. Joiner be awarded his special damages as are proved at trial plus an appropriate amount as general damages in an amount in excess of Ten Million Dollars ($10,000,000.00);

(d) Under Count Four, that Plaintiff Robert K. Joiner be awarded his special damages as are proved at trial plus an appropriate amount as general damages in an amount in excess of Ten Million Dollars ($10,000,000.00);

(e) Under Count Five, that Plaintiff Robert K. Joiner be awarded his special damages as are proved at trial plus an appropriate amount as general damages in an amount in excess of Ten Million Dollars ($10,000,000.00);

(f) Under Count Six, that Plaintiff Karen P. Joiner be awarded her special damages as are proved at trial plus an appropriate amount as general damages in an amount in excess of Ten Million Dollars ($10,000,000.00);

(g) Under Count Seven, that Plaintiff Robert K. Joiner be awarded his special damages as are proved at trial plus an appropriate amount as general damages in an amount in excess of Ten Million Dollars ($10,000,000.00);

(h) Under Count Eight, that Plaintiffs be awarded punitive damages in an amount in excess of Twenty Million Dollars ($20,000,000.00);

(i) That the cost of this action be levied against Defendants; and,

(j) That Plaintiffs be awarded such other and further relief as this Court deems just and proper.

Respectfully Submitted,
BURGE & WETTERMARK

By: /s/ Michael J. Warshauer
MICHAEL J. WARSHAUER
Georgia Bar Number 018720

Suite 3250, One Atlantic Center
1201 W. Peachtree Street, NE
Atlanta, Georgia 30309
(404) 875-2500

IN THE UNITED STATES DISTRICT COURT
FOR THE NORTHERN DICTRICT OF GEORGIA
ATLANTA DIVISION

(Title Omitted in Printing)

**ANSWER OF THE DEFENDANT,
MONSANTO COMPANY,
TO FIRST AMENDED COMPLAINT**

COMES NOW, MONSANTO COMPANY (hereinafter "Monsanto"), Defendant in the above-styled cause, and for Answer to Plaintiffs' First Amended Complaint, shows the following:

FIRST DEFENSE:

In answer to the numbered paragraphs of Plaintiffs' First Amended Complaint, Defendant MONSANTO says:

1.

This Defendant is without knowledge or information sufficient to form a belief as to the truth of the allegations contained in Paragraph One (1) of Plaintiffs' First Amended Complaint.

2.

This Defendant is without knowledge or information sufficient to form a belief as to the truth of the allegations contained in Paragraph Two (2) of Plaintiffs' First Amended Complaint.

3.

With respect to the allegations contained in Paragraph Three (3) of Plaintiffs' First Amended Complaint, this Defendant denies that MONSANTO has committed a tortious act in Georgia but admits the remaining allegations contained in Paragraph Three (3).

4.

MONSANTO admits that from time to time it manufactured polychlorinated biphenlys which were sold in bulk to General Electric Company, and Westinghouse Electric Corporation, among others, for use in fire resistant dielectric fluid in electrical transformers. Except as herein admitted, Monsanto denies the remaining allegations of Paragraph Four (4) of Plaintiffs' First Amended Complaint.

5.

The allegations contained in Paragraph Five (5) of Plaintiffs' First Amended Complaint are admitted.

6.

The allegations contained in Paragraph Six (6) of Plaintiffs' First Amended Complaint are denied.

7.

The allegations contained in Paragraph Seven (7) of Plaintiffs' First Amended Complaint are denied.

8.

The allegations contained in Paragraph Eight (8) of Plaintiffs' First Amended Complaint are denied.

9.

The allegations contained in Paragraph Nine (9) of Plaintiffs' First Amended Complaint are denied.

COUNT ONE
(STRICT LIABILITY AGAINST DEFENDANTS
G.E. AND WESTINGHOUSE)

10.

In response to the allegations contained in Paragraph Ten (10) of COUNT ONE of Plaintiffs' First Amended

Complaint, Defendant MONSANTO realleges, adopts and incorporates herein its answer to the allegations contained in Paragraphs 1 through 9.

11.

The allegations contained in Paragraph Eleven (11) of COUNT ONE of Plaintiffs' First Amended Complaint are not directed to this Defendant, and thus require no answer of the Defendant MONSANTO.

12.

The allegations contained in Paragraph Twelve (12) of COUNT ONE of Plaintiffs' First Amended Complaint are not directed to this Defendant, and thus require no answer of the Defendant MONSANTO.

COUNT TWO
(NEGLIGENCE AGAINST DEFENDANTS G.E. AND WESTINGHOUSE)

13.

In response to the allegations contained in Paragraph Thirteen (13) of COUNT TWO of Plaintiffs' First Amended Complaint, Defendant MONSANTO, realleges, adopts and incorporates herein its answers to the allegations contained in Paragraphs 1 through 12.

14.

The allegations contained in Paragraph Fourteen (14) of COUNT TWO of Plaintiffs' First Amended Complaint are not directed to this Defendant, and thus require no answer of the Defendant MONSANTO.

15.

The allegations contained in Paragraph Fifteen (15) of COUNT TWO of Plaintiffs' First Amended Complaint

are not directed to this Defendant, and thus require no answer of the Defendant MONSANTO.

COUNT THREE
(FRAUD AGAINST DEFENDANTS G.E. AND WESTINGHOUSE)

16.

In response to the allegations contained in Paragraph Sixteen (16) of COUNT THREE of Plaintiffs' First Amended Complaint, Defendant MONSANTO alleges, adopts and incorporates herein its answers to the allegations contained in Paragraphs 1 through 15.

17.

The allegations contained in Paragraph Seventeen (17) of COUNT THREE of Plaintiffs' First Amended Complaint are not directed to this Defendant, and thus require no answer of the Defendant MONSANTO.

18.

The allegations contained in Paragraph Eighteen (18) of COUNT THREE of Plaintiffs' First Amended Complaint are not directed to this Defendant, and thus require no answer of the Defendant MONSANTO.

19.

The allegations contained in Paragraph Nineteen (19) of COUNT THREE of Plaintiffs' First Amended Complaint are not directed to this Defendant, and thus require no answer of the Defendant MONSANTO.

20.

The allegations contained in Paragraph Twenty (20) of COUNT THREE of Plaintiffs' First Amended Complaint are not directed to this Defendant, and thus require no answer of the Defendant MONSANTO.

COUNT FOUR

21.

In response to the allegations contained in Paragraph Twenty-One (21) of COUNT FOUR of Plaintiffs First Amended Complaint, Defendant MONSANTO realleges, adopts and incorporates herein its answers to the allegations contained in Paragraphs 1 through 20.

22.

The allegations contained in Paragraph Twenty-Two (22) of COUNT FOUR of Plaintiffs' First Amended Complaint are denied.

23.

The allegations contained in Paragraph Twenty-Three (23) of COUNT FOUR of Plaintiffs' First Amended Complaint are denied.

COUNT FIVE

24.

In response to the allegations contained in Paragraph Twenty-Four (24) of COUNT FIVE of Plaintiffs' First Amended Complaint, Defendant MONSANTO realleges, adopts and incorporates herein its answers to the allegations contained in Paragraphs 1 through 23.

25.

The allegations contained in Paragraph Twenty-Five (25) of COUNT FIVE of Plaintiffs' First Amended Complaint are denied.

26.

The allegations contained in Paragraph Twenty-Six (26) of COUNT FIVE of Plaintiffs' First Amended Complaint are denied.

COUNT SIX

27.

In response to the allegations contained in Paragraph Twenty-Seven (27) of COUNT SIX of Plaintiffs' First Amended Complaint, Defendant MONSANTO realleges, adopts and incorporates herein its answers to the allegations contained in Paragraphs 1 through 26.

28.

This Defendant is without knowledge or information sufficient to form a belief as to the truth of the allegations contained in Paragraph Twenty-Eight (28) of COUNT SIX of Plaintiffs' First Amended Complaint and therefore the allegations are denied.

29.

The allegations contained in Paragraph Twenty-Nine (29) of COUNT SIX of Plaintiffs' First Amended Complaint are denied.

30.

The allegations contained in Paragraph Thirty (30) of COUNT SIX of Plaintiffs' First Amended Complaint are denied.

COUNT SEVEN

31.

In response to the allegations contained in Paragraph Thirty-One (31) of COUNT SEVEN of Plaintiffs' First Amended Complaint, Defendant MONSANTO realleges, adopts and incorporates herein its answers to the allegations contained in Paragraphs 1 through 30.

32.

The allegations contained in Paragraph Thirty-Two (32) of COUNT SEVEN of Plaintiffs' First Amended Complaint are denied.

33.

The allegations contained in Paragraph Thirty-Three (33) of COUNT SEVEN of Plaintiffs' First Amended Complaint are denied.

COUNT EIGHT

34.

In response to the allegations contained in Paragraph Thirty-Four (34) of COUNT EIGHT of Plaintiffs' First Amended Complaint, Defendant MONSANTO realleges, adopts and incorporates herein its answers to the allegations contained in Paragraphs 1 through 33.

35.

The allegations contained in Paragraph Thirty-Five (35) of COUNT EIGHT of Plaintiffs' First Amended Complaint are denied.

36.

The allegations contained in Paragraph Thirty-Six (36) of COUNT EIGHT of Plaintiffs' First Amended Complaint are denied.

SECOND DEFENSE

Any allegation in Plaintiffs' First Amended Complaint not specifically responded to in this Answer is herewith denied.

PREAMBLE TO AFFIRMATIVE DEFENSES

The Defendant MONSANTO reserves the right to rely upon the following defenses to claims asserted by Plaintiffs to the extent that such defenses are supported by information developed through discovery or by evidence at trial:

THIRD DEFENSE

Plaintiffs' Complaint fails to state a claim against Defendant MONSANTO upon which relief can be granted.

FOURTH DEFENSE

No act or omission on the part of the Defendant MONSANTO either proximately caused or contributed to any injury alleged to have been sustained by Plaintiffs.

FIFTH DEFENSE

Upon information and belief, this Defendant says that Plaintiff ROBERT K. JOINER assumed the risk of injury, if any he sustained, himself and therefore is not entitled to recover of this Defendant.

SIXTH DEFENSE

Defendant MONSANTO says that, even if it was negligent in the manner set out and charged in the First Amended Complaint, which alleged negligence is expressly denied, the negligence of Plaintiff ROBERT K. JOINER was equal to or greater than any negligence charged against this Defendant in such First Amended Complaint and the consequences of such negligence, if any in fact existed, could have been avoided had ROBERT K. JOINER been in the exercise of ordinary care.

SEVENTH DEFENSE

Upon information and belief, Plaintiff ROBERT K. JOINER has failed, in whole or in part, to mitigate and/or avoid any alleged injury purportedly sustained by Plaintiffs.

EIGHTH DEFENSE

Plaintiffs' claims for punitive damages are in violation of and barred by the due process clause of the 1983 Georgia Constitution, Article 1, Section 1, Paragraph 1, and by the Fifth and Fourteenth Amendments to the United States Constitution.

NINTH DEFENSE

Plaintiffs' claims for punitive damages are in violation of and barred by the equal protection clause of the 1983 Georgia Constitution, Article 1, Section 1, Paragraph 2, and by the Fourteenth Amendment to the United States Constitution.

TENTH DEFENSE

Upon information and belief, if Plaintiff ROBERT K. JOINER was exposed to polychlorinated biphenyls "PCBs") alleged to have been manufactured by MONSANTO and if Plaintiff ROBERT K. JOINER incurred any injury resulting therefrom, all of which is expressly denied, Plaintiff ROBERT K. JOINER's injury would not have occurred but for the fact that the "PCBs" were materially changed or altered by Plaintiff ROBERT K. JOINER or others, whether or not such others are parties to this action, and such material change or alteration was not made in accordance with the instructions of MONSANTO, was not made with the consent of MONSANTO or was not the result of conduct that reasonably should have been anticipated by MONSANTO, and therefore Plaintiff ROBERT K. JOINER's claim is barred.

ELEVENTH DEFENSE

Upon information and belief, if Plaintiff ROBERT K. JOINER sustained any injury or damage as alleged in the Complaint, which is denied, such injury was due to, caused by or contributed to, in whole or in part, to the negligence or carelessness of other parties over whom MONSANTO had no control and for whose conduct MONSANTO is not responsible. Such negligence of other parties was the sole proximate cause of, or an intervening, superseding, or insulating cause of the Plaintiffs' alleged damages.

TWELFTH DEFENSE

Upon information and belief, it is alleged that the Plaintiff ROBERT K. JOINER's employer at the time of the alleged exposure to "PCBs" was negligent in that the employer failed properly and safely to equip the Plaintiff ROBERT K. JOINER with the necessary protection; that the employer provided the orders and directions under which the Plaintiff ROBERT K. JOINER worked with or in the area of "PCBs" (if he did so); that the employer allowed "PCBs" to be used by the Plaintiff ROBERT K. JOINER and others in a manner so as to create a condition of danger for the Plaintiff ROBERT K. JOINER: that although the employer knew or should have known in the exercise of ordinary care of the general requirements of the handling of "PCBs", the employer nevertheless failed to relate all such information to the Plaintiff ROBERT K. JOINER; that the employer failed to provide a safe place for the Plaintiff ROBERT K. JOINER to work; that the employer failed to provide suitable training and education for its employees, including the Plaintiff ROBERT K. JOINER; that the employer failed to require the Plaintiff ROBERT K. JOINER and others to keep the premises and their persons clean and in a normal state of good housekeeping and cleanliness. If the Plaintiff ROBERT K. JOINER sustained any injury or damage as set forth in the Complaint, which is denied, the same was directly and proximately caused by the foregoing negligence of the Plaintiff ROBERT K. JOINER's employer and such negligence was the sole proximate cause of or an intervening, superseding or insulating cause of the Plaintiff ROBERT K. JOINER's injury and damage, if any.

THIRTEENTH DEFENSE

Any purchaser or user who purchased or used "PCBs" alleged to have been manufactured by MONSANTO, as the developers and manufacturers of the final product to

which the Plaintiff ROBERT K. JOINER was allegedly exposed, were sophisticated and knowledgeable users of "PCBs" and were in a far better position to instruct or warn Plaintiff ROBERT K. JOINER and, if any such instruction or warning was legally required, which is denied, their failure to instruct or warn the Plaintiff ROBERT K. JOINER was an intervening, superseding or insulating proximate cause of Plaintiff ROBERT K. JOINER's alleged injuries.

FOURTEENTH DEFENSE

If Plaintiff ROBERT K. JOINER was exposed to any "PCBs" alleged to have been manufactured by MONSANTO and if Plaintiff ROBERT K. JOINER incurred any injury resulting therefrom, all of which is expressly denied, Plaintiff ROBERT K. JOINER's claim is barred by the applicable statutes of limitations and/or repose.

WHEREFORE, the Defendant MONSANTO respectfully demands that judgment be rendered in its favor.

FREEMAN & HAWKINS

/s/ Joe C. Freeman, Jr.
JOE C. FREEMAN, JR.
GA State Bar No. 275700

/s/ Joanne Beauvoir Brown
JOANNE BEAUVOIR BROWN
GA State Bar No. 045730
Attorneys for Defendant
Monsanto Company

One Peachtree Center
303 Peachtree Street, N.E.
Atlanta, Georgia 30308
(404) 614-7400

IN THE UNITED STATES DISTRICT COURT
FOR THE NORTHERN DISTRICT OF GEORGIA
ATLANTA DIVISION

(Title Omitted in Printing)

ANSWER OF DEFENDANT WESTINGHOUSE ELECTRIC CORPORATION TO FIRST AMENDED COMPLAINT

COMES NOW defendant Westinghouse Electric Corporation and hereby raises its defenses and answers plaintiffs' first amended complaint (hereinafter "plaintiffs' complaint") as follows:

FIRST DEFENSE

Plaintiffs' complaint fails to state a claim upon which relief may be granted.

SECOND DEFENSE

Plaintiffs' complaint is barred by the statute of limitations.

THIRD DEFENSE

Plaintiffs' complaint is barred by the statute of repose.

FOURTH DEFENSE

Plaintiffs are barred from any recovery under the doctrine of assumption of risk.

FIFTH DEFENSE

Plaintiffs are barred from any recovery in whole or in part by the contributory negligence or comparative fault of plaintiffs.

SIXTH DEFENSE

The allegations of fraud set forth in count three, paragraphs 16-20, of the plaintiffs' complaint, are not stated with the requisite particularity.

SEVENTH DEFENSE

The plaintiffs have failed to specifically state items of special damages.

EIGHTH DEFENSE

Having raised its affirmative defenses and without waiving said defenses, defendant Westinghouse responds to each and every paragraph of the plaintiffs' complaint as follows:

1.

Defendant Westinghouse is without sufficient knowledge or information upon which to form a belief as to the truth or accuracy of the allegations contained in paragraph 1 of the plaintiffs' complaint.

2.

Defendant Westinghouse admits that it is a Pennsylvania corporation, that it maintains a registered agent in Fulton County, Georgia, and that it is subject to the jurisdiction and venue of this Court. Defendant Westinghouse denies that it commited a tortious act in Georgia as alleged in plaintiffs' complaint and denies all remaining allegations of paragraph 2 of the plaintiffs' complaint.

3.

Defendant Westinghouse is without sufficient knowledge or information upon which to form a belief as to the truth or accuracy of the allegations contained in paragraph 3 of the plaintiffs' complaint.

4.

Defendant Westinghouse admits that it purchased from Monsanto Company quantities of polychlorinated biphenyls for use in certain electrical transformers it manufactured. Defendent Westinghouse denies the remaining allegations of paragraph 4 of plaintiffs' complaint.

5.

Defendant Westinghouse admits that it was a manufacturer of electrical transformers which contained polychlorinated biphenyls and that some of these transformers contained polychlorinated biphenyls purchased from Monsanto Company. Defendant Westinghouse is without sufficient knowledge or information upon which to form a belief as to the truth or accuracy of the plaintiffs' allegations concerning General Electric Company.

6.

Defendant Westinghouse denies the allegations contained in paragraph 6 of the plaintiffs' complaint.

7.

Defendant Westinghouse denies the allegations contained in paragraph 7 of the plaintiffs' complaint.

8.

Defendant Westinghouse denies the allegations contained in paragraph 8 of the plaintiffs' complaint.

9.

Defendant Westinghouse denies the allegations contained in paragraph 9 of the plaintiffs' complaint.

10.

In response to paragraph 10 of the plaintiffs' complaint, defendant Westinghouse realleges and incorporates herein its responses to paragraphs 1 through 9 of the plaintiffs' complaint with the same force and effect as if they had been restated herein.

11.

Defendant Westinghouse denies the allegations contained in paragraph 11 of the plaintiffs' complaint.

12.

Defendant Westinghouse denies the allegations contained in paragraph 12 of the plaintiffs' complaint.

13.

In response to paragraph 13 of plaintiffs' complaint, defendant Westinghouse realleges and incorporates herein its responses to paragraphs 1 through 12 of the plaintiffs' complaint with the same force and effect as if they had been restated herein.

14.

Defendant Westinghouse denies the allegations contained in paragraph 14 of the plaintiffs' complaint.

15.

Defendant Westinghouse denies the allegations contained in paragraph 15 of the plaintiffs' complaint.

16.

In response to paragraph 16 of plaintiffs' complaint, defendant Westinghouse realleges and incorporates herein its responses to paragraphs 1 through 15 of the plaintiffs' complaint with the same force and effect as if they had been restated herein.

17.

Defendant Westinghouse denies the allegations contained in paragraph 17 of the plaintiffs' complaint.

18.

Defendant Westinghouse denies the allegations contained in pargraph 18 of the plaintiffs' complaint.

19.

Defendant Westinghouse denies the allegations contained in paragraph 19 of the plaintiffs' complaint.

20.

Defendant Westinghouse denies the allegations contained in paragraph 20 of the plaintiffs' complaint.

21.

In response to paragraph 21 of the plaintiffs' complaint, defendant Westinghouse realleges and incorporates herein its responses to paragraphs 1 through 20 of the plaintiffs' complaint with the same force and effect as if they had been restated herein.

22.

Defendant Westinghouse denies the allegations contained in paragraph 22 of the plaintiffs' complaint.

23.

Defendant Westinghouse denies the allegations contained in paragraph 23 of the plaintiffs' complaint.

24.

In response to paragraph 24 of plaintiffs' complaint, defendant Westinghouse realleges and incorporates herein its responses to paragraphs 1 through 23 of the plaintiffs' complaint with the same force and effect as if they had been restated herein.

25.

Defendant Westinghouse denies the allegations contained in paragraph 25 of the plaintiffs' complaint.

26.

Defendant Westinghouse denies the allegations contained in paragraph 26 of the plaintiffs' complaint.

27.

In response to paragraph 27 of plaintiffs' complaint, defendant Westinghouse realleges and incorporates herein its responses to paragraphs 1 through 26 of the plaintiffs' complaint with the same force and effect as if they had been restated herein.

28.

Defendant Westinghouse is without sufficient knowledge or information upon which to form a belief as to the truth or accuracy of the allegations contained in paragraph 28 of the plaintiffs' complaint.

29.

Defendant Westinghouse denies the allegations contained in paragraph 29 of the plaintiffs' complaint.

30.

Defendant Westinghouse denies the allegations contained in paragraph 30 of the plaintiffs' complaint.

31.

In response to paragraph 31 of plaintiffs' complaint, defendant Westinghouse realleges and incorporates herein its responses to paragraphs 1 through 30 of the plaintiffs' complaint with the same force and effect as if they had been restated herein.

32.

Defendant Westinghouse denies the allegations contained in paragraph 32 of the plaintiffs' complaint.

33.

Defendant Westinghouse denies the allegations contained in paragraph 33 of the plaintiffs' complaint.

34.

In response to paragraph 34 of plaintiffs' complaint, defendant Westinghouse realleges and incorporates herein its responses to paragraphs 1 through 33 of the plaintiffs' complaint with the same force and effect as if they had been restated herein.

35.

Defendant Westinghouse denies the allegations contained in paragraph 35 of the plaintiffs' complaint.

36.

Defendant Westinghouse denies the allegations contained in paragraph 36 of the plaintiffs' complaint.

WHEREFORE, having asserted its affirmative defenses and having answered plaintiffs' first amended complaint, defendant Westinghouse demands judgment that the plaintiffs' first amended complaint be dismissed with all costs cast against plaintiffs.

This the 2nd day of September, 1993.

/s/ David H. Flint
DAVID H. FLINT
Georgia Bar No. 264600
ALEX J. SIMMONS, JR.
Georgia Bar No. 646990
Attorneys for defendant
Westinghouse Electric Corp.

SCHREEDER, WHEELER & FLINT
1600 Candler Building
127 Peachtree Street, N.E.
Atlanta, Georgia 30303-1845
(404) 681-3450

IN THE UNITED STATES DISTRICT COURT
FOR THE NORTHERN DISTRICT OF GEORGIA
ATLANTA DIVISION

(Title Omitted in Printing)

ANSWER OF GENERAL ELECTRIC COMPANY TO FIRST AMENDED COMPLAINT

The defendant, General Electric Company (hereinafter "General Electric") hereby answers the First Amended Complaint as follows:

General Electric incorporates by reference and adopts its answer to the original complaint, specifically adopting and incorporating by reference in their entirety the First through the Fourteenth Defenses.

Additionally, General Electric answers the new Count Seven contained in the Amended Complaint as follows:

31.

Answering paragraph 31 of the First Amended Complaint, General Electric incorporates by reference its answers to paragraphs 1 through 30 of the First Amended Complaint as if restated verbatim.

32.

General Electric denies the allegations in paragraph 32 of the First Amended Complaint.

33.

General Electric denies the allegations in paragraph 33 of the First Amended Complaint.

As Count Seven in the original Complaint has now been characterized as Count Eight in the First Amended Complaint, General Electric incorporates by reference and adopts its answer to Count Seven from the original Complaint as though it were answering Count Eight in the First Amended Complaint.

WHEREFORE, having fully answered the First Amended Complaint, General Electric demands judgment in its favor as follows:

1) dismissing the First Amended Complaint with prejudice;

2) awarding all costs of litigation to General Electric; and

3) awarding all such other relief to General Electric as this Court deems proper.

This 3rd day of September, 1993.

Respectfully submitted,

/s/ Anthony L. Cochran
ANTHONY L. COCHRAN
Georgia Bar No. 172425

CHILIVIS & GRINDLER
3127 Maple Drive, N.E.
Atlanta, GA 30305
(404) 233-4171

[Filed Dec. 8, 1993]

IN THE UNITED STATES DISTRICT COURT
FOR THE NORTHERN DISTRICT OF GEORGIA
ATLANTA DIVISION

(Title Omitted in Printing)

DEFENDANTS' JOINT MOTION FOR SUMMARY JUDGMENT

The defendants, General Electric Company, Westinghouse Electric Corporation, and Monsanto Company, hereby jointly move for summary judgment in accordance with Rule 56 of the Federal Rules of Civil Procedure and Local Rule 220 on the ground that there is no genuine issue as to any material fact and that defendants are entitled to judgment as a matter of law. This motion is accompanied by a separate joint memorandum of law, as well as a separate and concise statement of material facts, all of which are filed contemporaneously with this joint motion.

Additionally, in accordance with Local Rule 220-5 (b)(3), the defendants are relying upon the following materials in support of their joint motion for summary judgment:

1. The deposition of Dr. Arnold Schecter, an expert witness retained by the plaintiffs;

2. Dr. Daniel Teitelbaum, a second expert witness retained by the plaintiffs;

3. Dr. Larry Robertson, a third expert witness retained by the plaintiffs;

4. The Declaration of Mary [*sic*—Mark] Homyk, an employee at The City of Thomasville Water & Light Department, Mr. Robert K. Joiner's employer;

5. The deposition transcript of the plaintiff, Robert K. Joiner.

6. The deposition transcript of Dr. Philip Cole, an epidemiologist retained by the defendants;

7. The deposition transcript of Dr. William Waddell, a toxicologist retained by the defendants;

8. The deposition transcript of Dr. William Bailey, a pulmonary expert retained by the defendants;

9. The deposition transcript of Dr. Thomas O. Rouse, a GE employee;

10. The deposition transcript of Dr. John E. Brown, Jr., a GE employee;

11. The deposition transcript of Dr. Stephen B. Hamilton, Jr., a GE employee;

12. The deposition transcript of Dr. George Frame, a GE employee;

13. The Declaration of Anthony L. Cochran, counsel for General Electric Company, concerning documents produced by the plaintiffs in this litigation;

14. The Declaration of Susan Van Vlack, a paralegal working with counsel for GE, concerning documents produced by the plaintiffs and The City of Thomasville Water & Light Department;

WHEREFORE General Electric Company, Westinghouse Electric Corporation, and Monsanto Company request that this motion be granted and that judgment be entered in their favor, dismissing with prejudice plaintiffs' complaint, with costs upon plaintiffs, and that defendants have such other and further relief as is just and proper.

This 8th day of December, 1993.

/s/ Anthony L. Cochran
(by ALC with
express permission)
ANTHONY L. COCHRAN
Georgia Bar No. 172425
Attorney for Defendant
General Electric Company

CHILIVIS & GRINDLER
3127 Maple Drive, N.E.
Atlanta, Georgia 30305
(404) 233-4171

/s/ David H. Flint
DAVID H. FLINT
Georgia Bar No. 264600
Attorney for Defendant
Westinghouse Electric
Corporation

SCHREEDER, WHEELER & FLINT
1600 Candler Building
Atlanta, Georgia 30303-1845
(404) 681-3450

/s/ Joe C. Freeman
(by ALC with
express permission)
JOE C. FREEMAN
Georgia Bar. No.
Attorney for Defendant
Monsanto Company

FREEMAN & HAWKINS
4000 One Peachtree
303 Peachtree Street, N.E.
Atlanta, Georgia 30308-3243
(404) 522-0856

[1]
IN THE UNITED STATES DISTRICT COURT
FOR THE NORTHERN DISTRICT OF GEORGIA
ATLANTA DIVISION

(Title Omitted in Printing)

A Deposition held at the Bache Building, Binghamton, New York, on the 14th day of July, 1993, commencing at 9:15 AM.

* * * *

ARNOLD J. SCHECTER, MD, having been called as a witness, being duly sworn, testified as follows:

* * * *

[20] Q. You mentioned a moment ago you reviewed [21] the adipose tissue test results. Did you at one point request that additional either adipose tissue or blood serum tests be run on Mr. Joiner?

A. I do not recall requesting that.

Q. Are you saying that you could have and you don't recall or you never did request them?

A. I just don't recall requesting them.

* * * *

[24] Q. In your standard practice, isn't it routine and customary for you to have blood tests run so that you can get a congener—congener specific level determined for dioxins, furans and PCBs?

A. No, it is not.

Q. To your knowledge, has another blood test for Mr. Joiner to determine dioxin levels actually [25] been done?

A. Not that I know of.

* * * *

[26] Q. Let me ask my question again, then. Do you have an opinion to a reasonable degree of medical certainty that, in fact, the chemotherapy and radiation

therapy that Robert Joiner underwent affected the level of PCBs, furans and dioxins in his adipose tissue?

[27] A. I would be surprised if there were not alterations, but I have no empirical evidence upon which to come to a conclusion.

* * *

[31] Q. Let's return to the results from Triangle Laboratories. You have seen those, have you not?

A. Yes.

Q. You say you saw those yesterday and you saw them also back when you were in Atlanta?

A. Yes, sir, that's correct.

Q. Do you recall what the total body burden was for Mr. Joiner reflected on those tests?

A. Yes, I do.

Q. What is your recollection?

A. For the most part, the dioxin and dibenzofuran levels were listed as nondetect or nondetectable. And the summation of the PCB congeners by the method employed by Triangle [32] Laboratory was roughly four-tenths of a part per million.

Q. How did you come to that four-tenths part per million?

A. Well, I think someone had simply added the PCB congeners and at the bottom of the page that's what they were as totaled.

Q. So, you didn't personally total them up?

A. No, I did not.

Q. Do you know whose handwriting that was that totaled them up at the bottom of the sheet?

A. No, I do not. I assume that coming up with 0.4 by simple addition is very simple and hard to make a mistake with, but I'd be happy to go over it with you if you'd like.

Q. Do you know if the results were reported in parts per million, parts were billion or parts were trillion?

A. The total that I recall for the PCB was four-tenths part per million and for the dioxins I believe it was in

parts per trillion. I believe it was lipid adjusted, but I'm not sure. It may not have been.

Q. Where did you meet Mr. and Mrs. Joiner?

[33] A. In Mr. Warshauer's office in Atlanta, Georgia.

Q. Why did you meet them?

A. I wanted to talk to them to get a feel for Mr. Joiner, to be able to have him describe to me what his exposure was in general, what his occupations had been like, what his chemical exposures might have been, what his medical history may have been to get a feel for what kind of person he was, whether he was a person I would find believable or not believable, to evaluate him and his cancer and to try to come to some conclusion as to the causation of his lung cancer.

Q. Was there any other reason for you to meet him?

A. Not that I can think of at this time.

Q. Now, you are an MD, is that correct?

A. Yes, sir.

Q. Did you physically examine Mr. Joiner?

A. I don't remember—I certainly did not do a complete medical exam or complete physical exam. I don't remember whether I did anything specific such as look at skin, I don't recall taking a blood pressure.

[34] Q. Did you take what some folks would refer to as your little black bag with you?

A. I don't recall doing that. But I could have and I might not have. I just don't remember at this time.

Q. Did you take his blood pressure?

A. I don't remember.

Q. Did you listen to his heart or lungs by means of a stethoscope?

A. I don't remember.

Q. Did you take his temperature?

A. No, I did not.

Q. Did you order any tests for him?

A. No, I did not.

Q. Why not?

A. Because he had a very thorough workup for his lung cancer and had been under intense medical care for a number of years when I saw him and I didn't want to put him through any more unnecessary hardship and I didn't feel the need for it.

Q. Did you take any notes during your meeting with Mr. Joiner?

A. I don't remember.

Q. You haven't presented any here today?

[35] A. If I have any, they are in my file and that was open to you yesterday and today.

Q. When you interview patients, do you typically take notes?

A. If I'm seeing someone as a doctor and they come to my office, and I'm seeing them for medical purposes, I take—I do a history and physical and make a note, yes.

Q. Did you do that for Mr. Joiner?

A. I did not feel I was seeing him as a regular patient. I was seeing him in an attorney's office on primarily a legal and medical legal matter.

* * * *

[36] Q. Other than that meeting in Atlanta and your meeting with Mr. Warshauer yesterday, have you had any other meetings face to face with Mr. Warshauer or the Joiners?

A. No, I did not.

* * * *

[38] Q. Other than Mr. Joiner's affidavit and his deposition transcript, and I believe you were also provided with a short three-line cigarette smoking history from Mr. Joiner. Other than those three documents, have you received anything else in writing describing Mr. Joiner's potential exposure history?

A. Not that I recall at this time.

Q. You made no notes that you recollect of your meeting with Mr. Joiner, is that correct?

A. Not that I recall at this time.

Q. Have you spoken with Mr. Joiner over the telephone at any time to ask him any questions?

A. Not that I remember at this time.

Q. So, have all of your communications, other than that face-to-face meeting, been with Mr. Warshauer or someone in his firm?

A. Yes, they have.

* * * *

[39] Q. You say Mr. Joiner was tired. You met with him just a few months ago?

A. Yes. I believe it was approximately a few months ago.

Q. This month is July of 1993, correct?

A. Yes, it is.

Q. So, you would have met with the Joiners in May or June?

A. I don't remember the date. Sometime this year, I believe. I don't recall the day.

Q. Was Mr. Joiner in remission when you saw him?

A. I don't know that I would use the term remission. He was a man who looked like he was going to be dead in less than a year. Whether his last cancer therapy had bought him a little extra time, perhaps it has, perhaps it hasn't. I don't know.

* * * *

[48] Q. Would it have been important to know whether other workers that he was around during the day smoked?

A. Well, it might have been of interest, but we had established that he had a smoking history for [49] many years and so did his parents.

Q. So, why would that have been significant to you?

A. Because most lung cancer in this country is related to cigarette smoking.

* * * *

Q. Setting aside the deposition for a moment, what do you recall he told you as an MD who was asking questions about his medical and smoking history?

[50] A. I recall that he came from a family of smokers. He smoked many years. He gave up smoking. He had other chemical exposures and he came down with lung cancer. That's how I saw it as an MD.

* * * *

[53] Q. Let me show you quickly what we've marked as Schecter Exhibit 10.

* * * *

Q. Did you assume that the social and personal history there given by Mr. Joiner to Dr. Morley was true?

A. With this is a brief note taken on a patient in acute pain with high temperature and is a very quick history. I have no reason to doubt that [54] he had a fever of 102 degrees Fa[h]renheit, small cell lung carcinoma, severe nausea, nasal discharge or he is allergic to Sulfa, Tylox and Codeine. And that his last chemotherapy ended on 1/92 for intensive treatment of small cell lung carcinoma stage III and that he remained disease free subsequently.

There is obviously a mistake in the family history which says negative for any evidence of carcinoma. The—that's clearly a mistake since we know his mother died of cancer. Positive for heart disease is correct. It states that the patient smoked one to two packs of cigarettes a day for 15 years. That seems a bit longer than I recall him saying he smoked, but I don't remember the year of starting or stopping. But two packs a day seems a bit higher. It's a quick history, a very short history done under acute illness, severe pain.

* * * *

[57] Q. His treating physician was Dr. Morley, one of them?

A. I presume so.

* * * *

[60] Q. Based on what you've learned in this case, what chemicals has Mr. Joiner been exposed to potentially?

A. What stands out after the cigarette smoke is the PCB transformer fluids and the PCB, dioxin and dibenzofuran contamination. And when I say dioxin and dibenzofuran, I'm referring to chemical contaminants or breakdown products which are frequently found in PCB fluids or in transformer fluids.

[61] Q. Any other chemicals that Mr. Joiner was exposed to?

A. Not that I know of or that I can recall at this time.

Q. Now, tell us what opinions you have reached in this case.

A. I believe more likely than not that Mr. Joiner's lung cancer was causally linked to cigarette smoking and PCB exposure. And by PCB exposure, I'm thinking also of PCBs and dioxins and dibenzofurans and related chemicals which frequently are found together in transformer fluids.

Q. Any-other opinion?

A. Well, that Mr. Joiner more likely than not has had a higher exposure or more exposure to transformer fluids and the chemicals in them than the average person, and this would include the PCBs, dioxins and dibenzofurans and related chemicals. And it seems to me from the history he's given, that until the mid-1980s, he was not aware of the fact that he was dealing with toxic chemicals and did not protect himself, nor did anyone attempt to protect him or inform him that he should be protected from these toxic chemicals. And this, of course, is a [62] breach of good occupational medicine or industrial hygiene practice.

Q. Any other opinions?
A. Not that occur to me at this minute.
Q. What do you mean by industrial hygiene practices?
A. I mean the practice by which workers are protected.
Q. By whom?
A. By government, by companies manufacturing toxic chemicals, by employers manufacturing toxic chemicals.
Q. Do you need any additional information in order for you to express these opinions?
A. Not that I can think of at this time.
Q. So, would these be considered your final opinions in this case?
A. Well, if there is further information that would alter them, then they might not be final. I don't hestitate in coming to these conclusions at this time. They seem to be reasonable.
Q. Do you plan any further investigation?
A. I haven't really thought about that.
Q. So, you've given no thought to any [63] additional investigation?
A. Well, I would think that if this isn't settled in a reasonable fashion and goes to trial, then one might want to be a little more thorough with Mr. Joiner, although I think I've covered the major basis more than adequately. And—
Q. You mean more thorough with his medical history?
A. Well, I might want to do a somewhat more thorough medical evaluations, although at this time, I really do not see the need for it. And I might want to visit the site of his employment and look at the transformers, look at the room involved, but I might want to examine medical [records] in more depth, although I don't see any point in examining the types of chemotherapeutic agents and their pharmacologic actions. I have sufficient knowledge for my purposes to know whether it would affect—know or not know as best anyone knows, how they might affect dioxin, dibenzofuran or PCB levels.

Q. Are you an analytical chemist?
A. No, I'm not.
Q. Do you know how to read a chromatogram?
A. No, I do not normally read chromatograms.

* * * *

[64] Q. Do you consider yourself an expert in oncology?
A. To the same extent that a Board certified physician in preventive medicine would be. No more, [65] no less for a licensed physician.
Q. You are not Board certified by any organization as an oncologist, are you?
A. As oncologist, no, I'm not.
Q. Ever practiced oncology?
A. No, I have not. In preventive medicine, we teach about the causes and prevention of cancer, but it is not my practice to see patients in oncology as an oncologist.
Q. Now, let's talk about your first opinion about the causal link. What facts do you base your opinion on that it's more likely than not that Robert Joiner's lung cancer is causally linked to cigarette smoking and exposure to PCBs and furans and dioxins, what facts is that opinion based on?
A. Most lung cancer in the United States is caused by cigarette smoking. And with cigarette smoking in the presence of other carcinogens, for example, asbestos or radiation, the rate of lung cancer will go up markedly. PCBs, dioxins and dibenzofurans are definitely promotors of cancer, that is, once a cell has been initiated, once the cancer has been initiated in the cell, then these chemicals definitely will promote the cancer. In [66] addition, in animal experiments by themselves, dioxins, dibenzofurans and PCBs cause cancer in a dose-dependent fashion. We can probably leave it there unless you want me to elaborate.
Q. Now, when you say that PCBs, dioxins and furans are definitely promoters, is that based on animal studies?

A. That these cause cancer is animal and human study. That they are promotors is based on in vivo and in vitro studies. The in vivo studies in animals show that these chemicals alone or following dosing with initiators of cancer will cause increase in cancers. The toxicity is also studying for these chemicals by a number of biochemicals means such as enzyme induction. And recent work such as that of Dr. George Lucier and colleagues at the National Institute of Health have shown that humans—human tissue appears to be as responsive as animal tissue to these chemicals, as laboratory rats, for example, which are sensitive species. The human studies are studies such as the Yusho poisoning where an excess of cancer has been shown in some studies. And Dr. Fingerhut's NIOSH study, some Seveso studies where humans were exposed to dioxins as published by [67] Dr. Bertazzi and others, IARC studies, International Agency on Cancer at the World Health Organization showing an increase in cancer in dioxin-exposed subjects and others.

Q. I want to go back. When you used the term promoter, are you talking about strictly animal studies?

A. These chemicals are considered a promoter based on evidence from laboratory studies.

Q. On animals?

A. On animals—primarily animal studies, yes.

Q. Whose studies are you relying on?

A. Many studies including that of Lucier and colleague. The studies I found, for example, many of them in the agency for toxic substances and disease registry toxicological profiles or PCBs and separate study or publication rather for dioxins and the separate publication for dibenzofurans where they've attempted to catalog the majority of the studies which strongly show that these chemicals are very powerful promoters.

Q. Again in animal studies, correct?

A. The—well, by animal studies, are you [68] excluding humans as animals?

Q. Yes.

A. You are refer to laboratory animals?

Q. Yes.

A. The studies for promoters per se have been primarily to date in animal studies. But human tissue has also been used for some studies of these chemicals and humans have been studied after exposure to the chemicals. But the difference is that we don't have the clear legally in this country to dose humans with toxic chemicals. The animal studies are far for powerful than the epidemiology studies where the sample size is smaller. We do not dose deliberately, so the dose is not known. The dose may be small. And latency periods cannot be followed as well as they can from animal studies or cell culture studies.

Q. Again, your opinion that PCBs, dioxins and furans operate as promoters is based upon studies of laboratory animals only, is that true?

A. The studies which tell us that PCBs, dioxins and dibenzofurans are promoters of cancer were developed through laboratory animals and we have belief that they are relevant to humans. We [69] test them on animals. We believe that's relevant to humans.

Q. Your opinion about PCBs, dioxins and furans being promoters is based upon studies of laboratory animals?

A. Yes. And my reason for believing this is relevant to human studies is because all government agencies concerned with health believe that this is the case and that we can usually pre-detect, because humans also are in the animal kingdom. Human cells are nuclei, cytoplasm, mitochodria, endoplasmic reticulum and respond in similar fashion.

Q. Object to the non-responsiveness to the answer after the word yes.

A. I would object to your keeping—stating that laboratory animals are different from human animals. I believe that we are in the same kingdom. That's what I've been taught. That's what I teach my medical students.

We do teach them that what is in animals is related to human.

Q. Object to the non-responsiveness.

Now, is it your opinion that cigarette smoking was the initiator of Robert Joiner's lung cancer?

[70] A. Well, that's an interesting question. We, of course, do not know whether the PCBs, dioxins and dibenzofurans also serve in an initiation function. One might—I would think that the cigarettes probably served as initiators and that the PCBs, dibenzofurans and dioxins probably more likely than not served as promoters of cancer. They may, in addition, also have served as initiators. These words aren't magical. They simply refer to whether the DNA is directly attacked or not.

* * * *

[71] Q. Based on your study in this area, what occurs when you administer the PCBs before the administration of the initiator in laboratory animal studies?

A. As I mentioned previously, PCBs alone also cause cancer, and this has been shown in many studies. They do not need to be—to have a promoter given first. So that PCBs, dioxins and dibenzofurans have been described as complete carcinogens by Dr. James Huff of the National Institute of Health because when given alone they will cause cancer to laboratory animals and probably to humans.

Q. Now, which chemicals are you speaking of there?

A. I'm speaking of dioxin and dioxin analogs which would include dibenzofurans, PCBs and other structurally very similar chemicals.

Q. Whose study was this?

A. The paper I just cited, and it was done by Dr. James Huff.

[72] Q. Did I understand you to say that this is all dose dependent?

A. These chemicals cause cancer in a dose dependent fashion.

Q. What is your understanding of the term dose dependent?

A. That means in simple language, the more you give, the more toxic outcome you get.

Q. Does it also mean that there is a threshold over which the chemical must reach in order to have any toxic effects?

A. No. As Dr. George Lucier and a Angelika Tritscher, George Clark and others have shown, a threshold is not implied nor is it necessarily found on biochemical responses with these chemicals.

* * * *

[75] Q. Let's move to your second opinion which was that in your opinion, Robert Joiner had higher exposure than the average person to [PCBs], dioxins and furans. On what facts do you base that opinion?

A. The fact that he worked with these chemicals.

Q. What other facts?

A. The fact that he worked with these chemicals for many years, he handled PCB contaminated transformer oil, he breathed smoke, he was in an underground enclosed area with no [76] protective gear for years. The rest of us do not normally handle chemicals contaminated with PCBs, dioxins or dibenzofurans as part of our work. That's not part of the work of the major population.

Q. What other facts do you base your opinion on?

A. Those are essentially the facts that I base them on, as I do normally in the practice of occupational medicine.

Q. Are there any other facts on which you can think of on which you base that opinion?

A. The documents we've discussed, my interviews with the patient and his wife, documents provided me by Mr. Warshauer and his colleagues.

Q. Now, your third opinion—

A. And I would perhaps even go a bit beyond that. I think it would be of less consideration than the others

opinion which usually a physician would come to the conclusion, the ones I've just given you. But the fact that this patient had a four-tenths—0.4 parts per million of total PCBs in his adipose tissue despite cancer chemotherapy, weight loss, weight gain after the exposure without protection and without customary protection against [77] toxic chemicals such as PCBs, dioxins and dibenzofurans, despite the fact that he still had 0.4 part per million of PCBs suggests to me that this is additional comformatory evidence for exposure beyond that of the average. Which we know he had because of his job.

Q. How many years did you assume Robert Joiner was exposed to PCBs, dioxins and furans?

A. I believe his exposure started on or about 1973, when I believe he started in the work, and continued at least until he began to wear protective gear and probably decreased in an amount—the protective gear was after that time in the mid-'80s.

Now, he may also, of course, have brought home these chemicals on his shoes and his clothes, as we know happens to workers exposed to chemicals. His clothes exposed to chemicals where we have the classical case of family members coming down with mesothelioma asbestos that the husband or father brought home on his clothes.

Q. Do you have any evidence to suggest that Mrs. Joiner has had any adverse health effects from PCBs, dioxins or furans?

[78] A. No. At this time, I have no evidence that that is the case. However, I believe it is probable that chemicals were brought to the home on shoes, on the clothing, on the skin of Mr. Joiner in those years when protective gear was not used and that there may be an increase in these chemicals in the home, or there may have been during those years.

Q. Looking at the affidavit of Robert Joiner, did you assume based on his affidavit that he would have had

roughly ten years of exposure without the use of any protective equipment?

A. Well, if he started in 1973 and somewhere after ten years began to wear protective equipment, I assume that he had exposure during those unprotected years.

Q. Now, during those unprotected years, how do you quantify his level of exposure?

A. There is no way of quantifying what has entered his lungs which is where he got the cancer and where it is the site of PCB metabolites. This is where PCB metabolites congregate and deposit. I assume that—it seems highly logical to me that he breathed PCBs, dibenzofurans and dioxins and so, there was an immediate passage through the lung [79] cells and to a certain extent deposition. We have no way of quantifying dose and target organ from that kind of exposure. There is no method that I know of published in the medical literature which would allow us to do that.

Q. So, you have no opinion as to the level of PCB, dioxins or furans in Mr. Joiner's lungs?

A. I have an opinion that it is above that which the general population would be exposed to.

Q. But again, you cannot quantify that, can you?

A. The exact amount, no, I cannot quantify the exact amount.

Q. What is the general background level for PCBs in the general population?

A. I'm sorry.

Q. What is the general background level in adipose tissue for PCBs?

A. In Americans?

Q. Yes.

A. The most recent findings I've seen are those of the Yosmeas which suggest that the levels in the last decade have gone down from roughly 1.2 parts per million in adipose tissue to roughly [80] 0.4 parts per million for the average adult in adipose tissue.

* * * *

[82] Q. In the United States in the last 20 years, have PCB fat levels usually been .5 to 1.5 part per million in fat for the general population?

A. Well, I don't know that the figures—just reviewed the EPA's PCB tissue levels in the last few months and that's not the data that was presented in their summary of adipose tissue levels for PCBs for the United States. I don't know what you are citing and I don't know whether that's correct or not correct.

Q. Now, what is the general background level in the United States for dioxins and furans in adipose tissue?

A. Are you asking for a specific congener?

Q. I'm asking for total, the accumulation of all of the congeners.

A. Total dioxins and dibenzofurans in adipose tissue, breast milk or blood on a lipid basis is roughly 12 to 1,400 parts per trillion.

Q. Do you recall what Robert Joiner's level was as revealed in the Triangle Laboratories data?

A. No, I don't recall the level.

* * * *

[83] Q. That does assume some form of actual intake, does it not?

A. Yes. Which everyone in the United States has. Or in any country I've ever tested people in so far in these industrial times.

Q. Is there any other published data that you use to determine background levels for the general population other than what you've already told us?

[84] A. My general knowledge of the field, attending meetings, reading the literature, talking to colleagues.

Q. Do you agree that if an individual's levels in fat tissue for furans, dioxins and PCBs are below background levels, it is more likely than not that the individual will not experience any adverse health effects?

A. No.

Q. What information do you have about the concentrations of PCBs, furans and dioxins in the mineral

oil contained in the transformers that Robert Joiner worked around?

A. We have specific data back that came by fax yesterday, and I showed you my entire file, showing that there were PCBs in some of the mineral oil. And my general knowledge that dibenzofurans and dioxins are found in PCB-containing electrical transformer oils.

Q. Now, you said some of the transformers. How many of the transformers that you have information on actually contained PCBs?

A. Some did and some didn't. I didn't attempt to add them up.

[85] Q. What was the level of concentration?

A. It varied from a few parts per million up to a few hundred parts per million in those that I looked at that came in yesterday.

Q. Do you recall seeing any that were greater than a few hundred parts per million?

A. I don't remember. I didn't look at every page.

Q. Did you see any record that indicated any of these transformers contained pure Aroclor?

A. No, I did not.

Q. From any source, do you have any information that any of these transformers that Robert Joiner worked on had pure Aroclor?

A. No, I do not.

Q. Do you have any information from any source that any of the transformers that Robert Joiner worked on had a concentration of Aroclor as high as 650,000 parts per million?

A. No, I did not.

Q. Now, the test certificates or the information that was sent to you yesterday, did it show testing for any substance other than PCBs or mineral oil?

[86] A. I didn't notice.

Q. Have you seen any information from any source that showed any content for dioxin or furans?

A. No. Not for these specific oils. I have, of course, for others.

* * * *

Q. Is it essential in the practice of medicine and in clinical research that a distinction be made between potential exposure to a toxic chemical on the one hand and actual intake of that chemical into the human body on the other hand?

A. That is a useful distinction.

Q. Why is it useful?

A. Because actual exposure can lead to health consequences whereas the potential for exposure without actual intake would not lead to a physical deterious effect.

Q. Isn't it also true that the fact that you [87] may be exposed to a chemical does not necessarily mean that you have taken it into your body?

A. Of course. But if a chemical is in the air, it's very hard not to breathe. I can choose whether I want to drink the coffee in the cup in front of me, but I cannot choose what's in the air that I may wish to breathe or not breathe.

Q. Is it possible to distinguish between potential exposure on the one hand and actual intake on the other hand by determining the tissue level of the toxic chemical in the fat in the exposed person and then comparing that level with those of the general population?

A. It sometimes can be useful. It's one medical test that one uses in coming to a medical conclusion, but one never should practice medicine based on one test alone. And it can also be not useful.

* * * *

[89] Q. Do you know Mr. Joiner's rate of [90] absorption to a reasonable degree of medical certainty?

A. I haven't thought about it for him. You are talking about PCBs or dioxins?

Q. All three.

A. I hadn't really thought about that up until this time. At this time, I have no opinion.

Q. Do you know to a reasonable degree of medical certainty what his metabolic rate of clearance is for PCBs, dioxins and furans?

A. Well, I don't think that information is available for anyone, let alone Mr. Joiner.

Q. To a reasonable degree of medical certainty, do you know his sensitivity, that is Robert Joiner's sensitivity, if any, to PCBs, dioxins and furans?

A. Yeah. We don't have any good methods of estimating genetic sensitivity. Except by the response such as enzyme induction or the formation of cancer which he has, so my conclusion was that these chemicals played a certain role in the promotion of his cancer and that he has a certain sensitivity to them.

Q. What is it, do you know?

[91] A. He is sensitive.

Q. How do you know that?

A. Most people are sensitive, most animals are sensitive. We have a voluminous data on human response in Yusho and Yuchem to humans exposed to PCBs, dibenzofurans and dioxins.

Q. Now, in those two incidents, the Yusho and the Yuchem, in both of those incidences, what was the concentration of PCBs, was it a 65 percent concentration, was it 650 parts per million Aroclor or some equivalent?

A. I don't remember what it was. It was PCB and dibenzofuran and dioxin contamination of rice oil which was used to cook food. I don't think anyone can be certain of what the intake was.

Q. Do you know of any animal studies that have ever been conducted by anyone where the test substance that was given to the animal had a dose of 500 parts per million PCB?

A. I don't recall what the dosage used in the very many studies of PCBs have been. You are referring to animal studies?

Q. Yes.

A. There would have been a huge number of [92] studies.

Q. In all of those studies, hasn't the dose that's been administered to the laboratory animal been far, far in excess of 500 parts per million, dramatically higher?

A. As I said, I don't have the doses memorized. What I can do would be to tell you that Peterson's recent study of central nervous system damage, reproductive damage and nerve damage even the smallest dose of two, three, seven, eight TCDD given to the mother caused damage to the offspring, brain damage, cognitive damage, emotional damage, hormone damage to the offspring. And I also know that in the Lucier, et al, experiments at the National Institute of Health, the biochemical end points they examined showed no sign of a threshold. The smaller the amount given, there was still a small response. It did not seem to taper off to 0.

Q. What was the dose of the two, three, seven, eight TCDD given in those studies?

A. I don't have it memorized. It's easy to look up.

Q. Now, you moved from PCBs to dioxin. Let's go back to PCBs. Do you know of any [93] studies—any laboratory animal studies where the dose that was administered was as little as 500 parts per million?

A. I do not have any of the doses memorized for the voluminous PCB animal and cell culture data. I don't have any of them memorized.

Q. As a research scientist, you don't have any idea whether the dose given to those laboratory animals is far, far in excess of 500 parts per million, you just don't know despite all of your reading over all of these years?

A. 500 parts per million doesn't tell you anything if you are given one drop versus one gallon of something. You are giving me a—one part or 500 parts in a million parts of something else. You are not telling me what the total dose is, so it's not a real number. You are not saying, Dr. Schecter, do any PCB animal studies use more than .6 picogram per kilogram per body weight given five days a week or seven days or week or given one time. You are asking the question in a very strange fashion and

I certainly have not memorized that. And in my library I have voluminous reviews of the PCB animal data. We can certainly do that if you [94] would like to do that this afternoon or tomorrow. I certainly make no attempt to memorize the many, many toxicology experiments that are relevant to this field.

Q. In any of the animal studies in which you base your opinion in this case, do you know what the dose was that was administered to the animals?

A. Do I have any memorized at this time, no. At this time, I have made no attempt to try to review or memorize the dose, including those where I have done part of the toxicology myself and have been a co-author on the studies. I think it's silly to say do you remember whether it was .2 picograms per kilograms or .8. I don't remember.

* * * *

[107] Q. Do the levels in human adipose tissue for higher chlorinated PCBs remain relatively constant for several years?

A. The total level or the congeners?

Q. The congeners.

A. Well, if you are asking did a higher PCB levels—are they excreted slower than lower ones, the answer is yes. If you are asking for specific congeners, I'm not sure I know the answer. And again, as a rule of thumb, I'm going to say yes.

* * * *

[112] Q. Now, are you aware of any animal studies that have been done on mice that would suggest that PCBs are a potent anti-carcinogenic agent?

[113] A. There are studies which would show—which found less tumors of a certain kind in animals exposed to PCBs or to dioxins than in controlled animals under the circumstances of the experiment.

Q. In other words, instead of being a promoter, it was an inhibitor?

A. That could be one interpretation. Another interpretation is that it's a statistical fluke. The sample size

was inadequate—this is something that's been considered and studied in many laboratories.

* * * *

[114] Q. Now, you mentioned back early on this morning about Mr. Joiner's work history, that there were lights that were used to heat the cores. Do you remember that testimony?

A. Yes, sir.

Q. All right. What temperatures are you assuming, for purposes of your opinion, those field lights caused the cores to reach?

A. Now, I'm not certain how hot the temperature was.

Q. So, you don't have an opinion?

A. No. I recall that he said the paint was blistered off, occasionally would turn red hot, but I don't know what that temperature is.

Q. Do you agree that laboratory studies have established that the paralysis of chlorobenzene at temperatures of at least 600 degrees centigrade with yield dioxins and furans?

[115] A. Yes. And they've also established it at lower temperatures.

Q. How much lower?

A. Well, that's a subject of debate. Incinerators seem to be producing it as the temperature goes lower on the way out. But that's certainly been well-documented.

Q. Do you agree that laboratory studies have studied that for paralysis of PCBs again you have to have temperatures of at least 600 degrees centigrade to produce furans?

A. No. Your statement is not correct.

Q. What minimum temperature is necessary?

A. I don't know the minimum temperature, but in 200 to 600 degree, you get a yield of PCBs being empirically converted to a reasonable larger yield of dibenzofurans and a somewhat smaller of chlorinated dioxins and the chlorinated benzenes of which you spoke

previously. Again, in the 200 to 600 degree centigrade temperature will produce a yield to greater or lesser extent of chlorinated dioxins and/or dibenzofurans.

* * * *

[118] Q. What's the minimum temperature there to create furans?

A. They are hot.

Q. What's the minimum temperature which they are produced?

A. Again, we don't know the lowest level.

Q. It's well above 200 degrees centigrade, isn't it, way above 200 degrees centigrade?

A. The question is at the hottest part you do try to keep the waste incinerators very hot to dry toxic chemicals. But as you go out of the flame, out of the heated area, you go through an area which is much cooler and there is a production of dioxins and dibenzofurans according to current research in the field.

Q. But do you know what that temperature is?

A. The lowest temperature, I don't know what the lowest temperature is.

* * * *

[127] Q. Was Mr. Joiner's level of dioxin found by Triangle Laboratory well below background levels?

A. Triangle Laboratory had a major problem for doing this. I give no credence to their results. They are not a World Health Organization. They did not take part in this round robin tests. And in any good laboratory result in human tissue, you should at least have a recognizable level for each of the 16 usually reported dioxins or dibenzofurans, and usually they had nondetect. Which to me indicates they had a great deal of trouble in laboratory methodology.

Q. Let me object to the responsiveness of the answer and ask the question again.

Wasn't the level of dioxin found in Robert Joiner's adipose tissue sample by Triangle Laboratories lower than the general background level? Either it was or wasn't.

MR. WARSHAUER: Object to the form of the question because it requires him to rely on a test result—
[128] A. My answer is garbage in, garbage out. They obviously have major problems with that laboratory analysis and I was a physician who is very experienced in that field and can give it no credence whatsoever.

Q. Was the number which was reported below background levels?

A. If the lab obviously had a major methodologic problem, I wouldn't use that in coming to a conclusion.

As we mentioned earlier, we described the numbers that I think are average for the United States, but that means you get results, you have a good lab doing good results. And as you know, the lab error for dioxin laboratories is subsequently generally about 25 to 40 percent plus or minus. But you should have a number. You shouldn't have nondetect.

Q. In your article in the Banbury report, did you publish a table that had the dioxin and dibenzofurans in whole blood from various countries in parts per trillion and it was recorded for the United States?

A. Well, yes, I did.

[129] Q. What did you report there for dioxin?

A. I reported—and again, we had only one nondetect. At an extremely low level of 16 or so congeners we looked at, we had a total of 1,591.

Q. What about for the furans?

A. I'm sorry. I thought you were asking me for both. I misspoke if you were—

Q. Dioxins?

A. For dioxins in this series, A pool of 100, I reported 1,499 parts per trillion in the lipid.

Q. What did you report for the furans?

A. And for the furans I reported 92 parts per trillion. But again, I must emphasize if I as a physician see a laboratory report that comes back to me obviously with a mistake made or no values, I do not treat a patient or evaluate a patient based on that. If a laboratory test is in error, you ask to have it repeated or you ignore it.

Q. Object to the non-responsiveness to the answer.

Do you know of any animal studies on respiratory effect in laboratory animals after inhalation exposure to PCBs, dioxins or furans?

[130] A. There are very few, if any, laboratory tests that have been done on animals with respect to respiratory toxicology of dioxins, dibenzofurans and PCBs. And I do not remember the content of the section on respiratory toxicology and the toxicologic profiles which I reviewed in the last year. I don't recall that at this point, if there [131] were any. I know that they are extremely sparce, if any.

* * * *

Q. Do you know of any animal studies on cancer in laboratory animals after inhalation of exposure of PCBs?

A. I don't recall any at this time.

* * * *

[133] Q. Does the dilution of PCBs in mineral oil reduce or inhibit the formation of furans if the mineral oil solution which has PCBs in it is heated? Do you know if the presence of the mineral oil inhibits or reduces the formation of furans?

A. I don't recall any studies that I have [134] read addressing that issue.

* * * *

[137] Q. Getting back to a line of questions that Mr. Cochran asked you earlier relating to PCBs, furans and dioxins contained in mineral oil or transformer fluid, you had given an analogy in answer to one of his questions about it being unknown at what point each of these substances—what the low end of the point of each one of these substances would vaporize. Do you remember that testimony?

[138] A. I don't think that's what I said. I think I said that on the vaporization question I didn't have the point memorized, but on the—in converting in heat and in the presence of oxygen in the PCBs converting to dibenzo mostly and dioxins, to a lesser extent, I believe that it's still subject to scientific research as to what the lowest temperature that will happen.

Q. Can you tell me at what point a PCB vaporizes?

A. No, sir. I haven't attempted to memorize that.

Q. Is it a very high number like 600 degrees centigrade?

A. That's the kind of thing I just look up when I need it. I just don't memorize that kind of data.

Q. It's well above the point that water boils, is it not?

A. I don't even try to memorize that data.

Q. Do you know what temperature water will vaporize?

A. I mean, sure, I would agree with you. First of all, I don't memorize temperatures, I don't [139] memorize numbers. I try not to do that unless it's something I use every day. If I can look it up in a couple of minutes, I'll look it up. It doesn't vaporize—it doesn't evaporate very easily. If that's the point you are getting at, I agree with you there.

* * * *

[143] [Q.] I'd like to know the percentage of time over the last ten years beginning at the year furthest away that you have spent seeing patients.

A. I don't know what that is. It would probably be less than 10 percent now and over the last ten years.

* * * *

[145] Q. Are you presently seeing patients?

A. I see a small number.

Q. How many, for example, are you going to see this week?

A. This week I'm not going to see any.

Q. Last week?

A. I didn't see any last week.

Q. The week before that?

A. Well, didn't see any the week before that.

Q. Did you see any during the month of May?

A. I think I saw several. I don't recall.

Q. When you say several, is that between five and ten or five and nine or four and nine or what?

A. Well, several means less than five to me.

Q. Do you recall what sort of malady or [146] condition the patients that you saw came to you for either examination or treatment?

A. I try to discourage patients in coming to see me except in any occupational problems. I try to expose chemical exposure other than the chemicals I'm interested in and those are PCB, dioxins, dibenzofurans, DDT.

Q. For each of the patients that you saw in May, were they for the purpose of evaluating what a particular chemical had done for them or had not done to them?

A. The last patient I saw were follow-up patients after chemical exposure.

Q. Were each of the patients that you saw involved in some sort of litigation or anticipated litigation?

A. The ones in May were.

* * * *

[150] Q. This morning I noticed that a check was delivered to you for, I think, $2,800 for today's time?

MR. COCHRAN: $2,600.

Q. $2,600 dollars for today's time?

A. That's correct.

* * * *

[161] Q. You count this as an eight-hour day?

A. This is a full day that's tied up.

Q. So that's about $325 an hour?

A. I didn't do it that way. But if you tell me that's what the arithmetic is, I certainly won't argue.

Q. What's your salary from the State University of New York?

A. $87,000 plus fringe benefits.

Q. If you work 24 days a year—25 days a year, that's 65,000 in litigation consulting fees?

A. With no fringe benefit, no pension, no health insurance, no sabbaticals and so on.

* * * *

[166] Q. How old is Mr. Joiner?

A. Approximately 39.

Q. So, if he smoked for 15 years and started when he was 18, that would make him 33. Which is not 1980, according to my calculations. So, does that change your opinion in any way?

A. I would say the conclusions—the conclusions that we discussed earlier?

Q. Yes.

A. As I recall, we had four conclusions. Can we run over them one at a time and do them one at a time because they may be different in one but not in the other and I just don't remember them at the moment.

Q. Opinion number one is that Mr. Joiner's lung cancer was caused by cigarette smoking and PCB exposure.

My notes may not be as exactly what you said, and then I've got including dioxins, furans and related chemicals.

A. I think my conclusion would be the same on that one.

* * * *

[168] Q. Your opinions do not change in any way whether he smoked for eight years or 15 years and whether he smoked one pack a day or two packs a day?

A. My conclusion is the same as what we just said a minute ago.

Q. Now, also, you said that he was exposed to more PCBs than the general population, do you remember saying that?

A. Yes.

Q. How much more?

A. Well, I told you that I don't think anyone can quantitate it and that I really don't think I or anyone else can quantitate it. I can say, it's in excess of what he would have consumed had he not taken it into his body if not for this job and this scenario that we discussed and it's more than the general population.

[169] Q. How much more?

A. I don't know.

Q. 1 percent more?

A. I don't know. I don't think anyone can come up with that and one of the big problems is you do not know how much got into the target organ because certainly he breathed some PCBs, dioxins and dibenzofurans from this job, but what the amount is is in question. But some of it went directly through the lung tissue to get into the bloodstream and the lung tissue is generally a target organ. PCBs—and specifically, he has personally come down with lung cancer.

Q. Are you saying that he came into contact to these PCBs through ingestion, dermal contact or breathing of vapors?

A. By ingestion, gastrointestinal tract?

Q. Yes.

A. It would seem to me that he ate food without necessarily cleaning his hands and the food almost certainly on some occasions was contaminated with an excess of PCBs and dioxins and dibenzofurans from his job. And number two, he breathed without protective gear. Heated and unheated transformer [170] oil which, in some cases was contaminated with PCBs and if PCBs, almost certainly dibenzofurans and dioxins. And third, he did not protect his skin from that, so his hands and face and neck and shoes and feet almost certainly got some transformer oil.

Q. You are saying all three?

A. Yes.

Q. How much, can you give us any idea of quantity? Is it 1 percent more than the general population or 100 percent more or how much?

A. I don't think it's scientifically possible to come up with a good estimate, and particularly with respect to the target organ that we're most concerned with, how much got into the lung.

Q. Now, you've also said that the Triangle Lab report which showed his body burden—

A. Which did not show his body burden.

Q. You said it was defective. How was that test ordered?

A. I have no idea.

Q. You said that as a scientific—when you see a test that you don't think is reliable, you repeat it or you ignore it?

[171] A. Well, as a physician. First of all, I happen to be one of the people, the physician scientist who develops this kind of testing in humans in America. And so, I have a fair amount of experience as to when this looks believable and when I tell a chemist, you guys have goofed.

Q. Did you repeat it or did you ignore it?

A. The dioxin, dibenzofuran portion of that test in my professional judgment is clearly in error.

Q. Did you repeat it or ignore it?

A. I didn't order the test. The test was ordered by a law firm.

Q. What law firm?

A. I presume it was your law firm (indicating).

Q. So, you ignored it?

A. I didn't ignore it. I just told the folks who had ordered it that I didn't think you ought to be relying on this for anything. The dioxin, dibenzofuran portion.

Q. But you did rely on it for purposes of the PCB level?

A. I came up for the conclusion to answer [172] certain questions I was asked this morning. That particular laboratory is well known for doing environmental testing, soil and so on.

* * * *

Q. You said the test showed four parts per million?

A. Four-tenths. This is from memory.

Q. This was in the range of what one would normally expect?

[173] A. This is what you would expect from an average North American population of healthy adults if done by the same technique that Yosmeas uses where he came up with the survey where his survey showed that the

PCB levels in fat tissue are decreasing and they are now down to by coincidence I think, it was approximately four-tenths part per million.

Q. The same shown in the test by Mr. Joiner?

A. Yes. But Mr. Joiner has lost a lot of weight and gained weight and has extremely traumatic chemotherapy and radiation therapy. My assumption is that his level of PCBs is diluted from what it was prior to the loss of weight and gaining of weight. And I also don't know what his level was prior to exposure to PCBs.

Q. I heard you say that, but I also heard you say that you don't have any studies or empirical evidence which show that there is a reduction in the PCB level caused by weight loss and subsequent weight gain or by chemotherapy.

A. I was referring to dioxin and dibenzofuran where is concern. And I said that the—

[174] Q Would you say PCB—

A. Also, let me finish the logic. In thinking that this four-tenths might possibly be in conjunction with the medical and exposure history evidence of higher than expected levels. He lost weight. He lost fat. Lost a large amount of fat. Fat has the highest concentration of PCBs, dioxins and dibenzofurans. These are fat soluble. If you lose fat, probably you are losing—and you lose it for a period of time, probably you are losing PCBs, dioxins and dibenzofurans. If you then regain from new food the fat and the weight, you are probably diluting.

Q. I heard you say that. My question was: You don't have any empirical evidence of that, you are just making that hypothesis, there is no evidence or proof of that of any kind?

A. Not entirely. For example, without any doubt that in nursing mothers, when the fat goes down due to nursing, the mother loses her fat due to giving it to the baby in the milk. We know that her dioxin, dibenzofuran and PCB level goes down as she nurses. And her levels are frequently found to be on average lower after nursing one baby and then the [175] second baby. That's been

published in the work of Furst. We do have evidence as to loss of fat, the dioxin and dibenzofuran and literature is not so clear. There is some old PCB data which suggests that maybe alteration—but because the PCB methodology has changed and also because I don't remember the details of all the published literature, I can't cite you anything that I would rely that has been published that I think is worthwhile to answer that question.

* * * *

[176] Q. Doctor, I just have a few more questions I'd like to ask following up about your scientific methodology.

As part of your opinion, are you assuming that the heating of the transformer cores created furans or dioxins, are you assuming that?

A. I'm assuming that where PCBs were present, and they were present in some of these cores, I believe there probably was a certain degree of formation of dibenzofurans and dibenzo—dioxins. I'm also assuming that in general, it's my understanding of what we know in the field that as PCB containing transformer fluids are used, that there is a certain chemical change of PCBs and an increase in dibenzofurans and to a lesser extent dioxins.

Q. But did I understand you to say you don't know the temperature that's necessary, the minimum temperature that's necessary to create furans and dioxins from the heating of PCBs, you don't know that number, do you?

A. I don't recall what the lowest published [177] number at this point is and as I told you, it's my understanding of the literature that it's still an active topic of scienific chemical-type research as to what the lowest level would be. I don't think it's so much in doubt that PCB containing transformer oils do usually contain dibenzofurans in normal. I think that's well established. I believe it's fairly well established that there is a gradual increase of the dibenzofurans in PCB containing transformer oil.

Q. And do you know what the temperature of that would have been created by the lights, you have no idea?

A. I don't remember whether it was discussed or I went over, but at this point, I don't recall what the temperature might have been.

* * * *

[178] Q. Did you assume there was any inhalation by Mr. Joiner of PCBs, furans and dioxins when the dielectric fluid was merely at room temperature?

A. Yes, I did, that there was some, yes.

Q. But again, you don't have any idea how much?

A. I cannot quantify nor do we know which congeners were necessarily present nor their relative toxicity because we don't have the chemical data. And we don't—certainly don't have any congener data from the environmental samples on PCBs, dibenzofurans or dioxins that I'm aware of. And one other point that you brought up, the dioxin toxic equivalent as they relate to PCBs. Many of them are still in question, so even if we had the [179] analytic data, there would be a certain amount of uncertainty as to the health consequences.

Q. As a general manner, do you agree that a fat biopsy will reflect not only the level at a given time, but will also reflect exposure for a certain period of time in the past depending on the congeners identified?

A. Would you please read that again a little slower?

Q. Do you agree that a fat biopsy, that is, one done on adipose tissue, will reflect not only the level at a given time, but will also reflect exposure for a certain period of time in the past depending upon the congeners identified for PCBs, dioxins and furans?

A. Well, it can. Although, I'm a little puzzled as to why you added the last phrase in your question. It was almost easier to answer before you added that, but it can. It can reflect under certain conditions and especially average absorption, average excretion, whatever that may be, correct time of sampling, excellent lab and a healthy adult patient.

* * * *

JOHN D. ARCHBOLD MEMORIAL HOSPITAL

Gordon Avenue at Mimosa Drive
Thomasville, Georgia 31792

HISTORY AND MEDICAL DR. MORLEY
Page 1

JOINER, ROBERT
MR# 235149 HOSP SVC: ONM
PT# 102095817 529 1

ADMITTED: May 10, 1992

CHIEF COMPLAINT: Mr. Joiner presented to the clinic with fever of 102 degrees Fahrenheit, small cell lung carcinoma, and severe nausea which has been present for several days. He has had significant nasal discharge with daily fevers, and presents for admission.

PAST MEDICAL HISTORY: He is allergic to Sulfa, Tylox and Codeine. He has had no major illnesses. Last chemotherapy ended in 1/92 for intensive treatment of small cell lung carcinoma stage III. He has remained disease free subsequently.

FAMILY HISTORY: Negative for any evidence of carcinoma. Positive for heart disease.

SOCIAL & PERSONAL HISTORY: Negative for cigarettes and alcohol use, although the patient did smoke 1-2 packs of cigarettes a day for 15 years.

PHYSICAL EXAMINATION: GENERAL: The patient is alert and oriented, but is complaining of severe nausea and vomiting, and is doubled-up because of abdominal cramping pain. NECK: Reveals no adenopathy or chyromegaly. HEENT: Reveals Horner's syndrome of left eye as before. The rest of the cranial nerves are essentially within normal limits. CHEST: Clear to ausculta-

tion and percussion bilaterally, although left lung sounds are decreased as previousy, because of surgical changes and radiation fibrosis. ABDOMEN: Diffusely nontender, with no organomegaly or masses, and normal bowel sounds. EXTREMITIES: With no clubbing, cyanosis or edema. LABORATORY: Pending.

IMPRESSION:

1. A patient with small cell lung carcinoma stage III, in remission.

PLAN:

The patient will be admitted to rule out recurrent disease. IV antibiotics will be administered because of fever, and fever work up will be obtained.

/s/ W. B. Morley
W. B. MORLEY, M.D.

RBM/plr

5/9/92 @ 5:48a
5/10/92 @ 4:17p

IN THE UNITED STATES DISTRICT COURT
FOR THE NORTHERN DISTRICT OF GEORGIA
ATLANTA DIVISION

(Title Omitted in Printing)

Deposition of DANIEL T. TEITELBAUM, taken on behalf of Defendant General Electric Company, pursuant to Notice of Taking Deposition in the above-entitled action, on Tuesday, October 12, 1993, at 10 a.m., at 3306 Independent Square, Jacksonville, Florida, before Shelli Kozachenko, CSR, RPR, and a Notary Public in and for the State of Florida at Large.

* * * *

[4] DANIEL T. TEITELBAUM,

having been produced and first duly sworn as a witness, testified as follows:

* * * *

[8] Q All right. Now, who prepared these, if you know?

A One was Joanne Milavec, and the other one was Nancy Fitzgerald.

Q All right. Who are Joanne Milavec and the other lady you just—Nancy Fitzgerald?

A They're two employees in my office.

Q And Joanne Milavec, then, is a paralegal?

A Yes.

Q Is she a full-time employee?

A Yes.

* * * *

[17] Q In reaching your opinion, are you assuming that there's been more than one fire?

A You don't know even what my opinion is, and I don't know what opinion you're referring to, so when you get around to it, why don't you ask me that question and I'll try to answer it for you. I really don't know what opinion you're talking about.

Q How many fires are you aware Mr. Joiner worked on, around, or near where the substance which caught fire contained PCBs?

A Well, Mr. Joiner describes in his deposition, and told me that in the early days of his career, he burned transformer oil, but he didn't keep a diary of [18] how many times, so I really wouldn't have any way of answering that question.

Q Okay. Anything other than that that you can recall—

A No.

Q —he told you about fires?

A I'd have to look at my notes and see what I have there. He did mention several occasions where there were transformer—

Q All right. Here are your notes. If you would—

A —fires.

Q —would you recount those for us, please? Would you mind if I came over there so I could read over your shoulder?

A Yes, I would mind. Let's make a copy, and then it will be easier.

Q Just put it here on the table between us, so we can read it—

A Well, I can't. I'd really—my eyes are such that it's just much more comfortable if I read it that way. Why don't we make a copy and then you can have a copy, and I can have a copy.

Q All right.

* * * *

[21] Q When you met with Mr. Joiner on July the 14th of 1993, did you conduct a physical examination of him?

A Yes.

Q What did you do?

A There's a notation of physical examination. I did as complete an examination as one could do not [22] working in one's own office, but actually there was pretty much everything that I needed, and so I did a typical examination just as—about the same as what I would have done had I had him in my own office, took his vital signs and went through and looked at him. I did not do any laboratory work, but I did enough of an examination to know what his condition was at the time that I saw him.

Q All right. You say you didn't do any laboratory work. Did you order any blood tests?

A No.

Q Any other diagnostic tests?

A No. He's had a very large amount of laboratory work, most of it quite recently. There was no reason to do that.

* * * *

[24] Q Have you seen a videotape of Mr. Joiner's work area?

A No, I actually didn't know about that until after I read his deposition, and I've not seen it, no. But it does correspond, from the description he gives in his deposition, to what he told me in his narrative history, apparently. Dr. Schecter describes it in some detail, and it sounds about the way it was described to me.

MR. COCHRAN: Object to the nonresponsiveness to the last half of the answer.

Q If you've never seen the videotape, how do you know that it corresponds to anything?

A Well, Dr. Schecter describes it. I don't know who took his deposition, whether it was you or not, but whoever it was went into a great deal of detail about it. And from the description which is there, it sounds very much like what Mr. Joiner told me his workplace looked like, so that's what I know about it.

Q Have you ever visited Thomasville?

A No.

[25] Q How many times have you met with Mr. Joiner?

A Just once.

Q Other than Mr. Joiner, have you had any conversations either face-to-face or over the telephone with anyone that has ever worked with Mr. Joiner?

A No.

Q Have you ever seen the analysis done by Triangle Laboratories?

A No, and the only thing I know about it is what Dr. Schecter discussed in his deposition.

Q Other than the adipose tissue sample that was tested at Triangle Laboratories, do you know of any other adipose tissue or blood samples taken from Mr. Joiner that have been tested?

A No.

* * * *

[27] Q Have you had any conversations about this case with a Dr. Julian Loube, L-o-u-b-e?

A Never heard of him.

THE WITNESS: What was that, L-o-u-b-e?

MR. WARSHAUER: B as in boy.

THE WITNESS: Just B, L-o-u-b?

MR. WARSHAUER: E.

THE WITNESS: Who was that?

MR. WARSHAUER: Just a treating physician in this case.

THE WITNESS: Okay. An M.D.? I'm sorry, I just wanted to make a note. It was not someone I'd ever heard of.

BY MR. COCHRAN:

Q Have you had any communications with any of [28] Dr.—or Mr. Joiner's, excuse me, treating physicians?

A No.

* * * *

Q All right. If you would, Dr. Teitelbaum, would you simply tell us what opinions you have reached in this case?

A Yes. I would say I have three opinions. The first opinion is that to the best of anyone's diagnostic capacity,

Mr. Joiner had lung cancer and has been treated and is currently in remission from that lung cancer.

Secondly, it's clear that for a period of many years, Mr. Joiner worked as an industrial electrician and had exposure to various materials commonly used in that trade, including mineral oils contaminated with PCBs.

And, three, that his lung cancer was caused by or contributed to in a significant degree by the materials with which he worked.

* * * *

[29] Q All right. Let's go to number two. Tell me each and every fact on which you base your opinion that he had exposure to mineral oil contaminated with PCBs.

* * * *

A Okay. In Mr. Joiner's history and in his deposition, he indicated that he began to work as an electrician for the City of Thomasville in June of 1973. He describes repeatedly in his deposition—if I could, can I have the medical chart? I need the . . .

Q (Tenders document.)

A In the form he completed which is indicated in my chart as occupational/environmental history form, he has circled all of the materials from the list that I present to each patient with which he worked, and he has handwritten PCB transformers as one of the items with which he worked.

[30] In his deposition he describes many years of work with mineral oil-filled transformers which he learned later in his career, following EPA investigations of the site, had been on occasions contaminated with varying concentrations of mineral oil—excuse me, with varying concentrations of PCBs.

He describes in exquisite detail, both in his narrative history to me and in his deposition, the many kinds of work that he did, including the draining of that oil, the transfer of the oil to various kinds of drums, the refilling of transformers, and the awareness which he developed

of the PCB contaminations after the EPA became involved in the control of PCBs.

He puts the date that he became aware of this as sometime between 1985 and 1987 when he stated that he attended an EPA class to learn about PCBs.

Q Where did he say that?

A On page 98 of his deposition.

Q All right. Go ahead.

A He states that sometime in that period, the city began to furnish gear, including Tyvek suits, overshoes, full-face respirators, and so on, but he himself—he stated there that he denied that that was because the EPA was pushing it. So I can't tell from the discussion what he actually knew, but he does state [31] specifically that he learned about PCBs when he went to the class.

So that's fundamental information that we have about his work history. There's nothing about this work history which is particularly different from many, many other electricians I've interviewed over the past 20 years who've worked in substations or in municipal distributing facilities.

I've seen people who've worked in Denver and in Colorado Springs and in Fort Collins and most of the cities in Western Nebraska and Eastern Colorado and many railroad substation electrical employees and so on. They all have about the same history. So I believe that those are the most important items.

Now, I understand that there are transformer analyses, but I don't know what dates they're from, so I can't give you any way of distributing those over the years in which he worked with those materials.

Q Do you know from any source what concentrations of PCBs were contained in any of the transformers on which he worked?

A I don't have those documents. I understand there are such documents; I have not seen them. Typically, in my experience, transformers which were listed as non-PCB transformers throughout this last 20 [32] years had

concentrations from anywhere above 50 parts per million to something under 1 percent, maybe 5,000 to 8,000 parts per million at the maximum.

Q. And is that something you're assuming for purposes of your opinion?

A Well, it's fairly typical of the era. I think it's reasonable to assume that that's so, yes.

Q All right. Do you know how long Mr. Joiner was exposed to mineral oil contaminated with PCBs, how many years?

A Well, he began to work in 1973, and he says he thinks he stopped sometime around 1987.

Q So you would be assuming a 14-year exposure?

A That's what he believes he was exposed—that's the period that he says. He says he does not know what the situation was after that.

Q In reaching your opinion, though, what length of time are you assuming?

A Well, that's the assumption that I have, because I have no history of his having worked with these materials before 1973. His medical history prior to that indicates that he worked—did a little farming, some farm chores early, and worked as a delivery boy and stocker—let me get the exact—baled hay and picked watermelon while he was in high [33] school up to 1972, and then in 1973 he began to work for the public utility.

Q Now, during the years that Mr. Joiner may have been exposed to mineral oil contaminated with PCBs, how often, if you know, was he exposed?

A I don't have, and have not seen that anyone has collected, the transformer records for who worked on them. I don't know how this utility works as far as its record maintenance is concerned, but other utilities that I've looked at keep a log for every transformer, and actually one could set up a record of how many he worked on, but how many hours it took him to do each job, it would be difficult to say. But from his descriptions to me, it was a very frequent activity.

* * * *

Q On the order of daily?

A I would say several times a week, not necessarily daily. It's more likely that there were periods that were quite intensive and went on for a number of days when he was working on a large transformer and baking it out, something of that sort, than that it was every day. So I think it was episodic but not every day.

Q Have you been shown any documents that would reflect the presence of dibenzofurans, dibenzodioxins, [34] or chlorinated benzene in any of the mineral oil on which Mr. Joiner worked on or around?

A I don't have any documents which would answer that question, There are, so far as I can tell, no quantitative analyses that have been supplied to me that answer how much and when.

* * * *

Q Or if any; is that correct?

A I don't have any documents at all. I think that one simply has to look at the likely chemistry of the situation and what's known about PCBs manufactured in this period and assume that there was some furan present, that there may have been some dioxin present, depending on the particular fire and circumstance. There probably was some chlorinated benzene since that's almost always a contaminant.

Q But that's speculation on your part in this case, is it not?

A No, it's not speculation. It's a reasonable assumption based upon what we know about the era. I cannot tell you what the specific concentrations are at any place or time. I mean, I think that could be answered by going to the supplier of the oil and getting their internal documents for the quality assurance on the materials, but I don't have such material.

* * * *

[37] Q Do you know when Mr. Joiner started smoking cigarettes?

A Well, I know what he says and what is recorded in the various medical records; there is some discrepancy among them. Around age 19. In the history which he completed for me, he said about 19 years was when he began to smoke cigarettes.

Q At age 19 or he smoked for 19 years?

A No, no. He says he—the question is, "How old were you when you began to smoke?" He says, "About 19 years."

Q All right. Does he indicate when he stopped smoking?

A Yes. It says, "How old were you when you [38] stopped smoking cigarettes regularly?" The answer is, "28 years," so I make that to be about nine years of cigarette smoking.

Q Did he indicate how many cigarettes he would smoke?

A Okay. The question I asked him was, "What was the usual number of cigarettes you smoked per day? Please give best estimate." And he said, "One pack per 24 hours."

* * * *

[39] Q Did you find any reference that he had smoked up to two packs a day for 15 years?

A I think I saw that somewhere.

Q Would that be 30 pack years?

A It would be, but that seems completely inconsistent with what everyone else found.

Q Do you know if that history was given by Mr. Joiner before he retained Mr. Warshauer?

A I really couldn't say. I don't know.

* * * *

[44] Q I want to make sure I've covered your second opinion. Have you, as far as you know, given us all of the facts on which you base that second opinion, that Mr. Joiner for many years had exposure to various mineral oils contaminated with PCBs?

A Well, I haven't talked at all about how he worked and the conditions under which he worked, all of which contributed to that.

Q All right. Tell me what facts you rely upon there.

A Well, for the first, at least, 12 to 13 years of his work he had no protective gear. He worked with [45] and in the oil on a regular basis. He describes frequently having his skin doused with oil. He describes needing a new pair of shoes every six months because of the oil destroying his shoes. He describes being inside the larger transformers on many occasions and being covered with oil. He describes inhaling the smoke on a number of occasions from baking out transformers.

He describes his desk as being in the area where the transformers were actually being serviced, so that—and his tools being constantly covered with oil. Again, this is very typical. Every electrician that I've ever talked to from this era told me that they used to use the oil from the transformers to oil their tools to prevent rust and to keep them in good shape. So he has multiple exposures by dermal and respiratory route.

Q Now, do you know how often, if at all, the mineral oil was contaminated with PCBs?

A No, I don't know that. As I said, I understand that there are documents, but I don't know from what era.

Q All right.

A I would say that from the era that we're talking about, virtually all of the mineral oil would [46] have been contaminated within the ranges that we're talking about. I think it would be unusual for it not to be contaminated at that time.

Q All right. So for purposes of your rendering your opinion, are you assuming that virtually all the mineral oil was contaminated with PCBs?

A Yes.

Q Okay.

A With concentrations between 50 and a few thousand parts per million during this era of time.

Q And that would be up to 1 percent?

A Up to 1 percent, yes. Generally speaking, I would say that in my experience the maximums have been around 6,000 parts per million, but as many have been 50 to 200 as have been 6,000.

Q And that's what you're assuming?

A. Yes.

Q Now, are there any other facts that you're relying on in rendering that second opinion concerning the many years that he would have been exposed to mineral oils contaminated with PCBs?

A I don't think I have any other information that would be helpful.

Q All right. Now, let's turn to the third opinion which was that—and I'm paraphrasing, so if [47] you want to go back—if I misphrase this, feel free to correct me, but as I understood it, your opinion is something like the lung cancer was caused by or contributed to in a significant degree by the materials with which he worked.

A Good notes.

Q All right. Now, tell me the facts on which you relied in reaching that opinion.

A Well, it's my opinion that cancer is a multifactorial disease. As a consequence of this multifactorial pattern of causation, various individuals respond with malignant disease at some time in their lives, and it is unusual for a single cause to be identifiable.

In my analysis of the information available concerning Mr. Joiner, I find three interesting factors. The first interesting factor is that there is at least one case of lung cancer in a first-order relative. There is not much evidence that sporadic cases of lung cancer have a familial pattern. There are recently some interesting studies on families of lung cancers, but they're pretty unusual. But there is at least one case in a smoker in his family. That's the first interesting thing.

The second interesting thing is that we do [48] know that Mr. Joiner smoked for a relatively significant period

of his life, but from the point of view of causation of lung cancer, a very—a low-end dose and early, and he stopped 12 years approximately before he got sick. But if the most recent studies on smoking and cancer are correct, our previous assumptions that as time passed the risk would disappear may have been unduly optimistic. So we have some risk of lung cancer because he was a smoker; however, the likelihood of his developing lung cancer at age 37 on a statistical basis is extremely small.

If you compare the lung cancer rates at age 60, which are around 400 per 100,000 person years, with the lung cancer rates in the 30s, which are almost too low to detect, somewhere around 10 to 15 per 100,000 and most of those in the later 30s rather than the early 30s, it's pretty—pretty low risk, but it's there.

The third thing is that we have a period of approximately 15 years of exposure to a material which, in and of itself, is an animal carcinogen, likely contaminated with additional materials which are animal carcinogens, carried in a vehicle which is a known carcinogen, and other work which he did which had some potential carcinogen exposure. It's not quite so clear, but he talks about using mineral oil as a—using [49] mineral spirits as a cleaning solvent.

So I see his occupational exposure to the materials containing—to mineral oil containing PCBs as a significant portion of his history. I am a firm believer in the concept that if there is more carcinogen exposure, there is more risk of cancer and more cancer in fact, and I believe that in that fashion, at least these three issues, some perhaps genetic predisposition, not well characterized, about which we know very little; smoking, about which we know more; and an occupational carcinogenic exposure to a material known to be an animal carcinogen and quite strongly suspected by most people being a human carcinogen as well, I think that those elements together are a significant portion of the causation of this disease.

Q Now, which of these substances is the known animal carcinogen?

A Well, I think that there's very little question that PCB is. There's sufficient information on PCBs. I brought the IPCS World Health Organization criterion because it's just hot off the press, and the summary which appears on page—let me find it—478 indicates that as of 1987, IARC had concluded that the evidence for carcinogenicity in laboratory animals is sufficient. This is the latest piece of information, [50] and there is no reason to doubt that, and they also concluded that PCBs are probably carcinogenic for humans.

Q Is it your opinion that Mr. Joiner's lung cancer was initiated by his cigarette smoking?

A I don't have an opinion about that. That would be purely speculative. It could have been. It could have been initiated by something entirely other than that. I think what's clear is that when you stop smoking, the promotional effect goes away, at least the nongenotoxic promotional effect.

Q The promotional effect of what?

A Of the cigarette smoking.

Q Is cigarete smoking a complete carcinogen?

A Hard to answer the question. I think there are components in cigarette smoke that have the potential for being complete carcinogens. For example, ethylene oxide is present. I think ethylene oxide can be both a promoter and an initiator. On the other hand, there are—and there are anthracenes and various polynuclear aromatics, all of which appear to have the capacity to be complete carcinogens.

On the other hand, there are a lot of materials which are only initiators, like urethane which is in cigarette smoke, or the tars which are probably [51] purely promoters. If you go back to the earliest research in cancer, the Rouses' work on chicken papilloma, the promoter used in those was tar, coal tar in that case but not terribly different from cigarette tar.

Q Let me ask you with respect to your opinion, in addition to cigarette smoke, mineral oil—well, let me start over.

In addition to the cigarette smoke and the PCBs, in your opinion, what other various chemicals were present in Mr. Joiner's workplace that you include in your opinion of—

A Well, I told you that there's mineral spirits. Mineral spirits is a very large kind of diffuse category of materials ranging from substances with flash points around 90 to substances with flash points of 150 to 200. If there —coal tar-derived mineral spirits, which there certainly are and they're still on the market in the United States, some of those may have had benzopyrene and other PNAHs in them. I think I would put mineral spirits in the mix as being a potential promoter and perhaps even an initiator.

Q Of Mr. Joiner's cancer?

A Yes. I think that—on the theory that cancer is multifactorial and is the result of all of [52] these influences working together in the right sequence at the right time, that's something that he did tell me about that I would add into the mix.

Q All right. Relative to mineral oil contaminated with PCBs, do you know whether Mr. Joiner was exposed to more mineral spirits or more mineral oil contaminated with PCBs?

A Oh, much more mineral oil—of the oil, much more.

Q What do you base that on?

A Because he said he occasionally used mineral spirits, and he was almost reg- —and he was regularly exposed to mineral oil. And the quantities were just enormously different. I mean, you're talking about a transformer that might have a couple hundred gallons of mineral oil in it and the use of a few gallons on a rare occasion of mineral spirits.

Q All right. Now, let me ask my question again. Relative to mineral oil contaminated with PCBs, do you know whether Mr. Joiner was exposed to more mineral spirits or mineral oil contaminated with PCBs?

A I think more mineral oil contaminated with PCBs—

Q And what—

A —by far.

[53] Q What do you base that opinion on?

A The history as he gives it to me and his deposition. I think you could answer that by looking at purchase records. I don't think that's a very obscure question, but I don't have those records.

Q Do you recall whether Mr. Joiner, each time they used a foot tub to move mineral oil, that they cleaned it with mineral spirits, each and every time?

A He didn't say each and every time. He said on some occasions. Sounds to me like they often didn't clean the tubs. It depends on whether they were using the tub to collect used oil that was going out, or if they were going to reclaim that oil, in which case they did try to clean it.

Q Okay. With respect to the filtering system that they used, did he tell you that they cleaned it with mineral spirits?

A On occasion.

Q And so you're assuming that he did not clean it each time.

A That's correct.

Q And you're assuming that they did not clean the foot tubs each time.

A Yes, and that seems to me to be consistent with his discussion.

* * * *

[54] Q Dr. Teitelbaum, let me ask you this: In taking the medical history from Mr. Joiner, did you make any determination about his exposure to secondhand cigarette smoke as a child?

A Yes.

Q And what did you determine there?

A Well, I determined—it's, I think, repeated in several things. His mother was a smoker at least, and he was certainly exposed to secondhand cigarette smoke as a child. What that amounts to is a debatable issue, but I would concede that he was exposed.

Q Do you know whether his father also smoked?

A I believe he did, but . . .

Q Do you know how much his mother and father smoked?

A No, I don't have that information, as I recall.

* * * *

[55] What significance to your opinions does it make that both his—if you assume both his mother and father smoked and were characterized as heavy smokers all during Mr. Joiner's childhood?

A Well, I don't know what to make of that. I mean, I have followed the EPA and the general scientific debate on sidestream smoke, and I think that the evidence is building that sidestream smoke probably is carcinogenic.

At the same time, I think that the dose of sidestream smoke is probably extremely small, and what's in that sidestream smoke is not at all clear, and it's probably changed over time depending on what tobacco was being burned, what paper was there, and so on. I would say it's part of the mix.

Q When you say "part of the mix," what do you mean?

A I explained to you how I think cancer arose in Mr. Joiner, and I think that his total life experience would be part of that. And the fact that his parents smoked would be part of that, but I would have no way of evaluating it and looking at the very large studies that [56] have been done. The risk for sidestream cancer—sidestream smoke-induced cancer is quite low. It's real, but it's very low.

* * * *

Q All right. In taking the family history from Mr. Joiner, did you determine whether any of his other relatives had been diagnosed with any forms of cancer?

A Let me go back and look at that because I don't want to speak out of turn. I think there was some discussion about one uncle who may have died of cancer.

Q Did you find any reference in the medical records to relatives other than his mother who had contracted some form of cancer?

A I believe so.

Q And what was in the medical records?

A Just hang on while I take a look a moment, try to tell you the answer. The 1990 record—1991 records from Dr. Morley indicated that his mother died of lung [57] cancer, two uncles died of unknown cancer, and various members have heart disease. The Archibold Hospital records from 5/92 show no family history of cancer, but other records agree with Dr. Morley's records.

Q All right. What significance is it to your opinion that, in addition to his mother, that one of his uncles had lung cancer and another uncle had some form of cancer which at this point is unknown?

A Nothing more than what I've said already.

* * * *

Q Well, let me ask you this: Turning to the exposure to his skin, do you know the amount of skin area that would have actually been exposed to mineral oil contaminated with PCBs?

A At various times I would say he was covered from head to foot, from his description. I think in general my discussions with many electricians, including [58] Mr. Joiner, is that the usual exposure is feet up to about the midcalf and arms up to the shoulders, because they would frequently work under the surface of the oil to make minor connections and corrections in the transformers. That would be pretty typical.

Q Do you know what the absorption rate through the skin is of PCBs that are contained in contaminated mineral oil?

A Well, I tried to answer that question. I can't find any data that anybody's studied that.

* * * *

[61] Q All right. In addition to mineral spirits, cigarette smoke, and PCBs, were there any other chemicals in Mr. Joiner's workplace that contribute to your opinion?

A In the occupational/environmental history which I took from him, he listed oil and paints as the only other two materials, the oil being something apparently other than the PCB oil, and the paints being apparently an occasional exposure. And I would think that the paints, if they're a problem, are a problem because they contain mineral spirits in some instances. That's all he indicated he worked with, so that's all I would point to. I don't have anything else.

* * * *

[65] Q All right. You've identified oil, you've identified paint, you've identified mineral spirits, you've identified PCBs. Is there any other chemical that was present in Mr. Joiner's workplace that you believe contributed to his lung cancer?

A Not according to anything he told me. I don't know of any other chemicals that were present, period. That's all we have.

* * * *

[67] Q Now, in reaching your opinion, did you assume that Mr. Joiner had any particular body burden of PCBs?

A Do you mean did I assume some quantity, no.

Q Why not?

[68] A Because I don't think that there is any way of relating the quantity of PCBs measured in anything with any outcome. All you can say is if you have more, you're more likely to have disease, and even that is not quite so clear when you begin to look at the studies.

Q Do you know what Mr. Joiner's body burden was as tested by the Triangle Laboratories?

A I've heard the numbers. I have no comment to make about it. I haven't seen—I know Triangle Laboratories well. They had their ups and downs over the years. With each specimen they provide a very extensive quality-assurance document. I have not seen that, and if I do see it, then I'd be able to make some evaluations as to whether I think the number is relevant or not relevant.

The number which was measured, as I recall, was about .5, but I don't know what to do with that because I don't know about the analysis that was done, and I don't know when it was done in relation to their other problems, whether it was before or after they were certified and so on. There are lots of things in that.

Q Do you know what the general background levels for PCBs and adipose tissue were in December of 1991?

A Where? And—

Q In the southeastern United States.

[69] A —by what method, and are you talking about total PCBs?

Q Yes, total PCBs for the southeastern United States.

A Okay. I'll allow—

Q Adipose tissue samples.

A I'll allow that in my mind when you talk about background, you're not talking about a normal level because a normal level would be zero. But the background, because of general environmental contamination, maybe is around .5 in fact, maybe. And I say maybe because there isn't a single definitive study that would answer that. They attempt to answer the question through what's called the National Human Adipose Tissue Study, NHATS. Really hasn't answered it.

The only year that's been studied is 1983, and it was kind of a trial balloon, and the data came out and was critiqued in many different ways and really is not to be considered reliable. So I don't know of any good study

that would answer that, but by impression from reading all of the studies which are around suggests it's around .5. But whether in Thomasville or Jacksonville or Fort Walton Beach they're the same or different from that, I don't know.

* * * *

[70] Q Can you say to a reasonable degree of medical probability whether Mr. Joiner's body burden of PCBs was higher than background nonoccupational levels before his diagnosis in August of '91?

A I have no opinion about that. It would be pure speculation.

Q Why would it be pure speculation?

A Because you don't have a measurement from before; you don't have any way of comparing it to the general population; you don't have anything that you could base that on. It would be absolutely a guess. The paper that's quoted in HSDB says that in their opinion, and it comes from—again, from a series of papers quoted in the EPA Drinking Water Criteria, that the burden of PCBs in human fat has been found to range between 500 and 1500 parts per billion.

Q Which would be .5 to 1.5 parts per million?

A That's right.

Q And that would be in adipose tissue?

A That's what it says, and that's from a paper [71] —that's from the U.S. EPA Drinking Water Criteria. On the other hand, in Michigan it's different, as you know, because of the PBB and PCB contamination from the Great Lakes. There's all kinds of numbers.

* * * *

[73] Q So you don't have any quantitative data to assess either exposure or dose here.

A That's correct. I would not have any way to do it. I don't have a reliable measure of PCBs in his body before he became ill. I don't have any reliable information concerning the contamination. I only know that in

the era we are talking about, the typical [74] materials were contaminated, as I've previously discussed, and he worked with it over a long period of time, and material is absorbed through the skin. That's as far as we can go.

* * * *

[75] Q Let me ask you this: Do you know the minimum temperature necessary to create furans or dioxins from the arcing pyrolysis or combustion of PCBs?

A You know, you asked that question of Dr. Schecter, and I thought it was—or whoever asked it, and I thought it was a very interesting question, and I tried to answer it. I didn't have at hand the kinds of chemistry and physics textbooks that would answer it. I don't know the answer to that, but it seems to me that that's a pure physical chemistry experiment, and that should be something pretty easy to answer in the laboratory.

I think we know the answer to that for, say, phosgene, its around 200 degrees C, and to create a more complex structure, it's probably somewhat higher than that. But I can't answer it now, and I didn't—I [76] couldn't get into the physical chemistry literature to get an answer to it, but it's an interesting question.

Q Mr. Warshauer didn't give you any of the analytical chemistry studies that have looked into that?

A Mr. Warshauer gave me what you have in front of you, and I think that he probably thought that was outside the scope of my expertise, as I would ordinarily think it is. But the question was interesting and I thought it worth looking up, but I couldn't answer it.

Q From the history that you obtained from Mr. Joiner or from any other source, were you able to determine the temperature created from the stadium lights that were used to bake the transformer coils?

A He says it was enough for it to smoke, and oil smokes at around 700 degrees, 800 degrees approximately.

Q Fahrenheit or contigrade?

A Centigrade. You'll get smoking at that.

Q Smoking of what, the mineral oil?

A Well, of the mineral oil or what's in the mineral oil. You'll get steam below that because there's water in it, but he describes it as real smoke, and I would say we're probably up around 700 degrees C.

Q And was that your assumption when reaching your opinion?

A About what?

[77] Q Is it relevant to your opinion, the temperature?

A It's relevant that when he heated the material, the more volatile substances would come off. It's not terribly relevant as to whether or not he was exposed. I think that the question you're asking is one that a physical chemist needs to answer, and I really can't—he describes it as being hot enough to smoke, and I think that's a sufficient piece of information for me.

Q You don't purport to be a physical chemist—

A Heavens, no.

Q —or an analytical chemist?

A No. I took physical chemistry and survived. I'm happy.

Q What kind of lung cancer does Mr. Joiner—did he have?

A Well, it's described as a small cell. But, again, that's a problem because he didn't have a total lung resection, and if you look at the cell type analyses that have been done in sophisticated studies, usually what you're hearing is a predominant cell, but if you do a large enough sampling, you'll find that there are more just the single type present.

* * * *

[78] Q Let's turn now to the literature, your bibliography.

A Okay.

Q I notice that you have some studies in there done by Lucy Anderson.

A Yes, hot off the press.

Q How many studies did she do that you have been able to identify?

A Well, I have, I think, four that I've looked at. I'm not sure they're all separate studies. I think they're interim reports on the same study or on the steps in the development of that study. The first [79] publication—let's see if I can—if I've got them right. I think the first publication that she put out was in 1983, and then in 1986 she did the single-dose study, and then in 1991/92 she published the promotion study with polynuclear aromatics, and then the 1993 publication is the single-dose study on infant mice on selective congeners. So I think what you're looking at is an ongoing work.

Q Was the promotion of lung cancer in the Lucy Anderson studies only for infant mice?

A I'll have to look and see. Was it "only" as distinct from any other kind of mice, or is that the only thing that she studied? What is it that you're asking me?

A Okay. I think I can answer your question.

Q Go ahead and answer the question.

A It appears to me that all that she studied were infant mice in the most recent publication, so that's as far as we can go. Whether the same thing would be present in other animals, I can't conclude from her study because she didn't give any information about [80] that.

Q Do you know if in the Lucy Anderson studies there was only one dose at which lung cancer was promoted?

A I'm not sure that her study answers that question. I think that that's—what you're asking is, is that the only dose at which, within the parameters of the experiment, she noticed that? I would say yes.

Q Okay.

A But whether the answer to your question on an absolute basis is that's the only dose at which it was promoted, I wouldn't be prepared to say that because she did not sacrifice over a period of time to answer that question.

Q Okay. Do you know of any other studies that you have located where somebody other than Lucy Anderson studied the promotion of cancer in animals by the administration of PCBs? Other than Lucy Anderson, anybody else, any other study that you found.

A A pure promotion study?

Q Yes, sir.

A Well, I can't say that I know all the studies, but I've read a lot. Most of them would not be sufficiently narrow to say that they studied a pure [81] promotional effect, so I don't think I can give you a study where the design of the study was to look at promotional events in whole animals.

I mean, you have all kinds of studies on the—on binding at the ah locus and so on, all of which contribute to the knowledge about promotion, but I don't think that there's another whole animal study which would—or another group that's doing whole animal studies that would answer that, at least that I've seen.

* * * *

[84] Q Well, if a chemical as a general matter is characterized as a promoter of cancer in humans, do you [85] classify it as a human carcinogen?

A It might be, but it is a chemical which participates in the generation of cancer. See, this is really a—it's a failure of the current terminology because our regulatory system and the scientific knowledge are not necessarily concordant. You surely don't want to go around exposing people to promoters.

Q Is that because they have a—as a general matter they would have a carcinogenic potential?

A No, it's because they would cause more cancer, but not because in and of themselves they're carcinogenic. And I think we're playing with words here. I think it's like saying if you need a detonator and dynamite together to get an explosion, is the detonator considered an explosive? And it may be for the Alcohol, Tobacco, and

Firearms Division, and you maybe would get charged with possession of explosives if all you had was the detonator in your hand, but that isn't going to blow up.

* * * . *

[87] Q Other than the studies by Lucy Anderson, do you know of any other animal study where lung cancer was promoted in a species other than mice?

A I don't think anybody's studied any other study. I think that now that the 1993 Anderson publication came out, I suspect you're going to see some more studies on that because now there seems to be a clean model that can be looked at.

[88] Q Is biologic plausibility a fundamental principle of toxicology?

A Yes.

Q All right.

A Well, no, wait. It's not a fundamental principle of toxicology, it's a fundamental principle of epidemiology, and it comes from Bradford Hill's criteria, and it is a fundamental principle which attempts to assess inference of causality based on scientific study.

Q Is it sufficient to establish biological plausibility to find carcinogenesis in only one animal species?

A It's a pretty good start. I mean, I think it is—

Q Is it sufficient?

A I think it's sufficient when you have all of the other information you have about PCBs as carcinogens. It's perhaps not sufficient to establish that a particular kind of cancer would occur in all animal species, but it certainly is much more consistent with the total body [sic] than if she had not found it.

If you came to me and you said, "Dr. Anderson has done this study and it's negative. Is that consistent?" I would say, "No, that's not consistent. [89] That's perhaps revolutionary." In fact, I would think that if you studied any kind of cancer well, you would find that it's probably—that PCBs are probably capable of promoting that cancer in a particular study design.

MR. COCHRAN: All right. Would you read that answer back to me, please, ma'am?

(The preceding answer was read by the reporter.)

THE WITNESS: Body of knowledge, I think, is what I said.

* * * *

Q Now, turning to your bibliography, I think we identified as Exhibit—is it 12A?

A Yes, right.

Q All right. What, if any, epidemiological studies have you reviewed in reaching your opinion?

A Well, I think I've reviewed all of those that are around on PCBs. I think that the study which in the long run will give us the most information is probably Bertazzi's group. I just saw Professor Bertazzi in Nice and I asked him if he has anything new coming out, and he says yes, he has something new coming out, but he wouldn't tell me what it was, so I don't know what it [90] is. Apparently, they've done some further study of this group of capacitor workers.

He could have another five years of observation, which would be a lot of person years. I think that's the best study. You've got Kuratsane's studies from Japan. You've got a few cases of lung cancer there. They're not very convincing, as the Japanese lifestyle is different. There's—it's, again, suggestive but not convincing.

I think that the recent review by IARC of all of the studies, including Gustavsson, who was also at the meeting, and Hogstedt, who was also there, and—all of whom talked about this, I think everybody is willing now to take the position that PCBs are human carcinogens, but I don't think that they would—any of them would be willing to say that they have evidence, convincing evidence, that PCBs cause any single kind of cancer.

I think they would say that they are human carcinogens and that the evidence now is that they can promote a whole series of cancers: hematologic, kidney cancer, certainly lung cancer is on the list. They've been observed

and they appear to be out—by Bertazzi's work, well out of proportion to what would be expected.

Q Okay. You said you have reviewed all of the [91] epidemiologic studies. Are there others that you have reviewed that are not on that list?

A These primarily were the ones that dealt with—that mentioned lung cancer and so on. Yes, there's a whole series of them listed in that WHO document, and I've—whenever one comes out, I collect it and I read it.

Q Do all of the epidemiological studies show the same thing; that is, are they consistent?

A No, but—all do not, but there is certainly general agreement that for groups which have been studied long enough and for which the exposure is well enough characized, there is an increased risk of cancer in humans. But you have a very varied group. You have groups where you have mostly women exposed; you have groups where you have mostly men.

Most of the studies come from capacitor workers, not from people who did this kind of—the work that Mr. Joiner has done. I don't know of a study in industrial electricians. Be interesting to do, but I don't know of such a study. And probably it would be one you could do because a lot of these people belong to the unions and would be fairly easy to identify.

Q Did the Bertazzi study have a very short follow-up period?

[92] A Yes, that's why I said he—he published it in '87, and I think he wrote it about—sometime in '86. We could theoretically have another five or seven years of follow-up, and he says he has something in the works, but wouldn't tell me anything about it.

Q If an epidemiologically [*sic*] study has a short follow-up period, does that put a limitation on the interpretation of the results?

A Can put a lot of limitations. If you're dealing with something which is very highly penetrant, a disease which occurs quickly and very often, like if you study leukemia in radiation victims, and you've got five years of follow-up,

it's clear in five years that you're going to have an increase in leukemia in people exposed to radiation.

On the other hand, where you're dealing with something which is a relatively weak carcinogen and a relatively rare disease—I mean, if you wanted to study PCBs in electricians at age 35, I haven't done the calculations, but you'd need millions, I think, millions of exposed people to get a statistically significant finding, or millions of person years anyway.

Q What was the size of the cohort that Dr. Bertazzi studied?

A I think 1500 approximately. Mostly women, I [93] don't remember, or half women, half men. I'd have to look back.

Q So would that, again, having a small cohort, be a limitation on his results?

A Got to wait longer.

Q Well, in addition to waiting longer—I mean, I take it you're saying you have to wait longer because you have such a small cohort?

A Well, you need a number of person years of exposure and a number of person years of follow-up. You can get the number of person years of follow-up by having a big group for a short time or a small group for a long period of time. A thousand person years of follow-up standardized over, they may give you an answer comparing studies, but—

Q Then would the study be inconclusive due to low statistical power?

A That's one of the accusations that have been made, and he himself says that they—you know, that the lung cancer occurrence was tending or trending towards statistically significant, but was not statistically significant.

That doesn't really mean anything, as you well know, because statistical significance is not the only criterion for interpreting information. It suggests [94] that there may be an error in the study, but if it's biologically plausible and it's consistent with other studies, even though it

doesn't reach statistical significance, you'd best be very, very cautious in saying the effect isn't there.

Q Did Dr. Bertazzi correct for confounding factors like smoking?

A He didn't because he didn't have that data. He may have that data now.

Q He did not?

A He did not, no. But he had a lot of women, and women are not big smokers apparently, he says, in Italy.

Q Was there a confounding factor involving the local rate of incidence?

A Well, there was a problem—there's the issue of whether he should have used local—a local comparison group or a national comparison group. I think the best of the two is really neither. I think you have a real problem with how you do that because, you see, if you choose the local comparison group to calculate your standard mortality ratios, the people who died locally already include in the denominator all the people who may be included in this study, okay? Everybody who died of lung cancer is going to be [95] included in the denominator, and it's going to include the people who were exposed here. So you're going to need a larger group in the numerator in order to reach statistical significance. So he said, "Okay, I'm going to go in the national levels."

Well, in the national level, you don't know how many of those people were exposed to PCB either. There's a paper by Alan Feinstein—Alvin Feinstein called "Fraud and Deceit in Epidemiologic Studies" in which he talks about inadvertent fraud based on death certificate studies, and he deals with all of this issue. And he says that the time has come to stop using death certificates because you have this kind of fallacy built into the study, and it tends to move the study towards the null and eliminate statistical significance.

That's why I said previously that the simple fact that it did not reach statistical significance isn't enough to convince me the study is not right.

Q All right. Let me ask you: Based upon the epidemiologic studies that you've seen, is there a finding of a significantly increased incidence of cancers of a general type in an exposed population when compared to an unexposed population in a carefully documented epidemiologic study?

A I think that's a subject for a doctoral paper, [96] and I'm going to say it's too vague for me to answer. You're going to have to tell me what you mean. Are we talking about all cancers? Are we talking about general populations? I think that the question you're asking me now has been answered by the expert commitee that IARC had together, and their conclusion was there's enough evidence to say that it's probably a human carcinogen, there is not enough evidence to say that is proved, and I think I would have no problem accepting that.

Q And when you say that it's a human carcinogen, you're just talking about generally without respect to any particular—

A That it's capable of causing cancer in humans, without saying which cancers it's capable of causing. I do not believe that there's any good evidence that PCBs in any specie have a signal lesion. I mean, we have a whole series of cancers which have been observed as increased: kidney cancer, lung cancer, hematopoietic cancers. I've even seen one study on prostate cancer. Lots of opportunities.

* * * *

[101] Q All right. As far as you know, does this Defendant's Exhibit 7 contain an accurate listing of the cases in which you've testified either at trial, deposition, or hearing from approximately May of 1983 through approximately January of 1992?

A I can't say how accurate it is. It's the best list that we could put together at the time, and since we no longer maintain it and I don't have anything to document this and can't reach it before 1988, I can't [102] tell you how accurate it is. After 1988, it's pretty good.

Q All right. And you say "we," when you say "we prepared it," are you talking about the folks in your office?

A Well, I was at Denver Clinic at the time it was originally prepared, and it was prepared by somebody at the clinic based on the records that they had. But I don't have access to them; they've gone bankrupt. I have no way of confirming anything before '88.

Q All right. But with respect to after 1988, it appears to be fairly accurate.

A Reasonably accurate. We got asked for it often enough so that we just made a list, and we add to it as something happens.

Q All right. And so you've reviewed this from time to time, then.

A Yes, roughly.

Q All right. Now, do you know how many times you testified in calendar year 1990?

A I don't know. Whatever's here is what there is. You can count them as well as I can.

Q Would 57 be a correct—

A Whatever it is, it is. That's how we made the list.

[103] Q All right. Since 1991—excuse me, since 1990, have you continued to testify with the same frequency that you testified in calendar year 1990?

A Well, you've got the list here through 1992, and so whatever there is here is what I've done. Actually 1990, I think, was probably a year in which I had more appearances, but most of them were very small issues, and I think that there have been less issues that I've testified on since that time. But probably 30 to 40 times per year would be a reasonable guess.

* * * *

Q Okay. You indicated we had it through calendar year 1992. Actually, if you look at the list, it only goes through January 14th of 1992, doesn't it?

A Oh, I don't know, whatever you have. I mean, I'm surprised you don't have more. Mr. Freeman's firm

should have a more recent one than that because they took my deposition in a case and asked for this. Ask Mr. Parnell, he probably has it.

* * * *

[104] Q Let me ask you, looking at these numerous cases here that you testified in in calendar year 1991, in how many of those cases, going down that list, did you testify for the plaintiff?

[105] A I haven't counted for that year, but it's almost always, I would say, for the plaintiff. Let's see for '91. In '91 I appeared for Eli Lilly as the defendant in Wyatt-Jose, and in—I think that was the only defense case I appeared in that year. That's no reflection of what work I do; that's only what cases actually went to some deposition or—

Q Well, let's—

A —trial.

Q —look at the 57 cases in 1990. How many of those, looking at the list, did you testify for a defendant?

A No, please, it's not 57 cases, it's 57 appearances, and sometimes it's repeated appearances in the same case. But I haven't looked at this one, so I don't know. I haven't seen this list in a long time.

Morrison versus Eli Lilly, I appeared for Eli Lilly. Sorrells, I appeared for Merck. Mullens, appeared for Wyeth. I think that's all.

Q So of the 57 appearances in 1993, three were for defendants?

A Three for defendants, that's correct. Usually I see the people who are sick, and usually if I feel that the causation is what I think it is and I write that in a report and somebody comes to me and says, [106] "Will you testify about that?" I will say yes. If I've told it to the patient, I'll tell it to the Court as well.

* * * *

Q Now, how much time each year do you spend in the United States?

A About nine months.

Q So these appearances that you have would take place during the remaining nine months of the year.

A Right.

Q So how often do you testify?

A By deposition?

Q Or court appearance or at a hearing.

A In court, not more than three or four times a year, maybe five times a year maximum, some years not at all. I think in '90—was it '91 or '90, there wasn't a single court appearance. By deposition, perhaps three or four times a month.

Q All right. And what do you charge?

A Depends. You have my correspondence file; it's got all the fees laid out. The current fee is $4,000 a day, $2500 for a half day, $500 per hour.

Q When you came to Atlanta to examine Mr. Joiner [107] back in July, how much did that cost?

A Half day, I believe. I was there for something else and saw him in the morning. I charged him for half a day.

Q $2500?

A Right.

Q And for your appearance today, it's another $4,000?

A That's correct.

Q Now, how long have you charged those rates, $4,000 for a full day and $2500 for a half day?

A About 1989, something like that.

Q So if you had 57 appearances in 1990 at $4,000 each—

A No, that's not correct. You're making an assumption.

Q All right. What would it have been?

A I have no idea, because many of them are an hour. I mean, you look at a Worker's Compensation case, and in Colorado we don't try it on paper as you do in many states. You have to give a deposition or appear at a hearing and those take an hour, they don't take a day or a half a day, so I have no way of knowing.

If you want to know what my total income is in a particular year, I can't give you that, but I can give [108] you an estimate. And you have to remember that I have 16 employees, so we're not talking about just one person's money in the pocket.

Having said that, I can't answer your question because I don't know for 1990, but I can give you a general idea of what my income is if that's what you want to know.

Q All right. What is your income annually simply from testifying?

A Well, I don't get paid that way. I get a salary like anybody else gets a salary. I have a corporation and a lot of employees and a building and very high overhead, and I get paid a salary like anybody else does.

Q What's your salary?

A I get—my after-tax monthly salary is $11,000.

Q What percentage of your income is attributable to involvement in litigation?

A I can't answer that either. I can tell you that 70 percent of my income comes from my consulting and 30 percent comes from patient care, although my time is 60/40.

Q All right. When you say consulting, is that consulting with attorneys who are involved in [109] litigation?

A It's everything I do in consulting. I consult with companies, I consult with attorneys, I consult with government agencies. Anything that I do on a case that's—anything other than—other than patient care or teaching would be included in that.

* * * *

[110] Q Now to be specific to this case, is there any study that you know of anywhere in humans that indicate that small-cell, being specific to this diagnosis, lung cancer was caused by PCBs?

A No. It's an impossible research question to investigate, I think, in the first place, and in the second place, there is no such study.

Q I see.

A So I think it's—in other words, I think one exists at the present time.

[111] Q I see. The same question with respect to PCBs as contributing to small-cell lung cancer in humans.

A That, I think, could be done, but has not been done.

Q It has not been done; is that correct?

A That's correct.

Q And there is no study of small-cell lung cancer that indicates that it was promoted in humans, is there?

A No, there is no such study. But let me comment on the fact that if you ask about small-cell cancer because of Mr. Joiner's diagnosis, I have pointed out that we do not have more than a limited sample, and that at the very first question which Mr. Cochran asked me, I pointed out that I am satisfied that to the best of the ability of the physicians who were treating him to say today that's what his diagnosis is, but I would not accept that that is, in fact, what his diagnosis is in the absence of a better tissue diagnosis.

Q Now, can you point to any study in animals where the person conducting the study has suggested that the animal developed small-cell lung cancer?

A I don't think there's an analogy in animals that would be relevant, so I can't point to such a study.

* * * *

[112] Q . . . As I understand it, you work for a corporation?

A I have what's called a professional corporation. You can call it that.

Q What is the name of your corporation?

A Daniel T. Teitelbaum, M.D., Inc.

Q And you own 100 percent of the stock in the corporation?

A Yes, that's correct.

[113] Q And that's where you receive your income?

A Yes.

Q And you have 16 employees?

A That's correct.

Q And do they all work there in the Denver office?

A Yes.

Q And I understood earlier that you—at least one of them is a paralegal?

A That's correct.

* * * *

[115] Q Well, you say it's none of my business, but you've charged me $4,000 in advance to take your deposition. So far I've been here three hours and 15 minutes—

A I'll be happy to give you a refund from 1:30 on.

* * * *

[117] Q Does your office have a brochure that you use for advertising purposes?

A Never advertised.

Q You do not advertise of any kind?

A The last thing in the world I'd need would be to advertise. I'd drown.

* * * *

[118] Q Now, you referred to this book, and I think specifically this book is called *The International Program on Chemical Safety, Environmental Health Criterion 140, PCBs, Second Edition.*

A Right.

Q And you referred, I believe, to page 478.

A Right, the summary page.

Q 477, which is paragraph—all right, 478 which is the first—let me see if I can find this. First paragraph, which says that PCBs were evaluated by IARC in 1977 and 1987. In 1987 the IARC concluded that because the role of impurities in PCBs and the carcinogenicity could not be excluded and because of the lack of knowledge on dose response relationships, the evidence from epidemiological studies is limited. However, the evidence of carcinogenicity in laboratory animals is sufficient, taking the combined evidence from human and experimental

animal studies, the IARC group concluded that PCBs are probably carcinogenic for humans.

A Right.

Q And you agree with that statement?

A Yes, I do.

[119] Q On the preceding page in paragraph 926, appraisal, it says that some epidemiological studies on occupationally exposed workers—and is that Yushall—

A Yusho, yes.

Q Yusho patients indicate an association between PCB exposure and cancer especially with regard to hepatobiliary—

A Yes.

Q —tumors; however, no definite conclusions can be drawn from available data because of the small number of deaths in the population studies, the lack of clear dose response relationships in the occupational studies, and the difficulty of evaluating the effects of other compounds present in PCBs.

A Yes.

Q Do you agree with that?

A Yes.

* * * *

MR. COCHRAN: Let's go back on the record. Can we agree and stipulate that what we're now going to mark as 7A is a more up-to-date list of the legal appearances by Dr. Daniel T. Teitelbaum?

MR. WARSHAUER: Subject to the restrictions of his prior testimony as to how it's prepared.

* * * *

[Teitelbaum Ex. 11-A]

JOHN D. ARCHBOLD MEMORIAL HOSPITAL

Gordon Avenue at Mimosa Drive
Thomasville, Georgia 31792

HISTORY AND PHYSICAL DR. MORLEY
Page 1

JOINER, ROBERT
MR# 235149 HOSP SVC: ONM
PT# 102093895

ADMITTED: May 03, 1992

PRESENT ILLNESS: Mr. Joiner presents to clinic with fever and shortness of breath, significant nasal discharge with daily fevers.

PAST MEDICAL H ISTORY: Allergic to sulfa, Tylox and codeine. Has had no major illnesses. Last chemo ended on January 1992 for intensive treatment of small cell lung carcinoma stage III. He has remained disease free subsequently.

FAMILY HISTORY: Negative for any evidence of carcinoma, positive for heart disease.

SOCIAL & PERSONAL HISTORY: Negative for cigarettes and alcohol use although the patient did smoke one to two packs a day for 15 years.

PHYSICAL EXAMINATION: The patient is alert and oriented, but is complaining of severe nausea and vomiting, doubled up because of abdominal cramping, pain. Neck reveals no adenopathy or thyromegaly. HEENT reveals Horner Syndrome of left eye as before. The rest of the cranial nerves are essentially within normal limits. CHEST: Clear to auscultation and percussion bilaterally although left lung sounds are decreased as previously because of surgical changes and radiation fibrosis. ABDOMEN: Diffusely nontender. No organomegaly or masses

and normal bowel sounds. EXTREMITIES with no clubbing, cyanosis or edema. LABORATORY: Pending.

IMPRESSION:

1) Patient with small cell lung cancer in stage III.

PLAN:

The patient admitted to rule out recurrent disease, IV antibiotics administered because of fever.

/s/ Walter B. Morley
WALTER B. MORLEY, M.D.

RBM/sm

6/21/92 @ 1:26p
6/21/92 @ 3:10p

[Defendant's Exhibit 7]

LEGAL APPEARANCES BY DR. DANIEL T. TEITELBAUM

CALENDAR YEAR 1983

DATE	CASE	TYPE OF APPEARANCE
1983	Jones v. Penwalt	Trial Testimony
05/18/83	Perry v. Summers	Trial Testimony
05/26/83	Charlesworth v. Davies	Deposition
08/04/83	Gouge v. Celestial Seasoning	Work Comp. Hearing
08/15/83	Sierra v. Sunstrand	Work Comp. Hearing
08/22/85	Ramon Lewis v. Aerojet General	Work Comp. Hearing
10/15/83	Amon v. Manville	Deposition
11/01/83	Pettis v. Boston Hospital	Trial Testimony
11/14/83	Martin Adams v. Granite	Work Comp. Hearing
11/09/85	Schwerin v. Cyanamid	Trial Testimony
11/83	People v. Templeman	Trial Testimony

CALENDAR YEAR 1984

01/11/84	Manly v. Zaki	Trial Testimony
01/12/84	Galloway v. Denver Metal Finishing	Work Comp. Hearing
01/25/84	Ruiz v. Chloride Battery	Deposition
02/01/84	Richner v.	Work Comp. Deposition
02/02/84	People v. Hobart & Steed	Trial Testimony
03/05/84	Jim McDuffie v. Burl. No. Railroad	Work Comp. Deposition
04/18/84	People v. Mary Hoffner	Trial Testimony
04/24/84	Agent Orange Plaintiffs v. Dow	Deposition
05/08/84	Schledowitz v. Schering	Deposition
05/14/84	Tejeda v. Eagle Chemical	Deposition
05/21/84	Skyring v. Wyeth	Deposition
06/11/84	Jourdan v. Syntex	Deposition
06/14/84	Broussard et al., v. Velsicol	Deposition
06/18/84	Gann v. Schwartz, et al.	Deposition
07/05/84	People v. Hoffner	Trial Testimony
07/10/84	Katsounakis v. Robertson Assoc.	Work Comp. Hearing
07/11/84	People v. Houston	Trial Testimony

DATE	CASE	TYPE OF APPEARANCE
07/23/84	Deasy v. U.S.	Deposition
07/24/84	Hunter v. International Harvester	Work Comp. Deposition
07/25/84	Pierce v. Anaheim Drug	Deposition
08/01/84	Popick, et al. v. Manville, et al.	Deposition
08/07/84	Genette v. Syntex	Deposition
10/02/84	George Maddox v. Colo. Ute	Work Comp. Hearing
10/10/84	Wallace Anderson v. Manville	Deposition
11/19/84	In RE: Jay F. Morris, D.D.S.	Administrative Hearing
12/05/84	Schmunk v. Merck, et al.	Deposition
	CALENDAR YEAR 1985	
01/08/85	Skeen v. Monsanto	Deposition
01/12/85	Clausen, et al. v. Texaco	Deposition
02/15/85	Esquibel v. Vail Med. Center	Deposition
02/21/85	Gaan v. Schwartz, et al.	Trial Testimony
03/22/85	Kast v. Upjohn	Deposition
04/03/85	Sturdy v. Hansen, et al.	Deposition
04/15/85	Hickman v. Thompson Company	Deposition
05/14/85	Kruse v. Ralston Purina	Trial Testimony
05/24/85	Ashley v. Scientific Applications	Trial Testimony
05/28/85	Deasy v. U.S.	Trial Testimony
06/04/85	Broussard et al. v. Velsicol	Trial Testimony
06/18/85	Russell v. Coacher	Deposition
06/19/85	Glaser v. Ashland Oil, et al.	Deposition
07/03/85	Daly v. Scheared, et al.	Deposition
07/19/85	Sauter v. Chestnut	Deposition
08/05/85	Durham v. Western Paving	Work Comp. Deposition
09/06/85	Blessing v. Dow	Deposition
09/10/85	Carman v. Sherwin Williams	Deposition
09/30/85	Donald Ward v. City of Mitchell	Deposition
10/02/85	Kerr v. State Hospital, et al.	Deposition
10/11/85	Hinojos v. O'Dell	Deposition
11/07/85	People v. Goode	Trial Testimony
12/06/85	Henderson v. Texaco	Deposition
	CALENDAR YEAR 1986	
01/06/86	Lancaster v. Newberry	Deposition
01/06/86	O'Neill v. Zoecon	Deposition
01/22/86	Jason Enos	Deposition
02/13/86	James Baisley	Deposition

DATE	CASE	TYPE OF APPEARANCE
03/04/86	Simon, Ledford, Duhon	Deposition
03/11/86	Summers	Deposition
03/27/86	Grenier v. Dow	Deposition
03/14/86	Formaldehyde Hearings	Hearing
04/03/86	Benzene Hearings	Hearing
05/16/86	Brown v. Snodgrass	Deposition
05/23/86	Sharon Lee	Deposition
06/23/86	Kast v. Upjohn	Trial Testimony
06/26/86	Burke v. Continental Can	Deposition
07/02/86	Robideaux	Deposition
07/17/86	Pierce v. Anaheim, et al.	Trial Testimony
07/22/86	Kitzman	Trial Testimony
08/05/86	Diamond v. Dilly	Deposition
08/06/86	Cook, et al. v. Fashion Carpets	Deposition
08/07/86	Charles Wright	Deposition
08/08/86	Kevin Goode	Deposition
08/08/86	Pete Zamara	Deposition
09/04/86	Nelson v. Scott's Pest Control	Deposition
09/09/86	Charles Wright v. Gulf Oil, et al.	Deposition
09/17/86	Hickman v. Thompson Co.	Deposition
09/22/86	Leslie Payne	Deposition
09/23/86	John Colcord	Deposition
09/29/86	House v. Aeroplane Club	Deposition
10/02/86	State v. Aoki	Trial Testimony
10/15/86	Geist v. Coors	Deposition
10/17/86	Baisley	Trial Testimony
10/21/86	Skeen v. Monsanto	Trial Testimony
10/28/86	Plaintiffs v. Union Pacific	Deposition
10/29/86	Roberta Sorensen	Trial Testimony
11/07/86	Bittle v. Dahlberg	Deposition
11/24/86	Brancucci & Langarek	Deposition
11/26/86	Robert Thompson	Deposition
12/15/86	David and Catherine Wells	Deposition
12/16/86	Griffin	Deposition
12/19/86	Bittle v. Dahlberg	Deposition
	CALENDAR YEAR 1987	
01/03/87	Ziegler v. Gulf States	Deposition
01/09/87	Summers	Deposition
01/30/87	Hofmeister	Deposition
02/09/87	Francis Larrew	Deposition
02/13/87	Cassens v. Upjohn	Deposition
02/13/87	Ronald Reef	Deposition
02/24/87	Burke v. Continental Can	Deposition
03/02/87	Runge v. Hecker, Ming Dynasty	Deposition
03/12/87	Stout	Deposition

DATE	CASE	TYPE OF APPEARANCE
03/24/87	Laboratory Standards	Hearing
03/25/87	Greene v. ARCO	Deposition
03/31/87	Lloyd Wright	Deposition
04/06/87	Colorado v. Idarado	Deposition
04/20/87	Scott v. Monsanto	Deposition
04/24/87	Snapka v. Big Red	Deposition
04/29/87	Barbara Nemec	Trial Testimony
05/11/87	Raymond Swanson	Deposition
05/12/87	James Sipes/Velma Porter	Deposition
05/21/87	Sargent et al.	Deposition
05/27/87	Paaka v. Coors	Trial Testimony
05/28/87	Jaspar Wages	Deposition
05/30/87	Southard	Deposition
06/03/87	Colorado v. Idarado	Trial Testimony
06/04/87	James Sipes/Velma Porter	Trial Testimony
06/05/87	Abernathy v. Mopac	Deposition
06/10/87	Snapka v. Big Red	Trial Testimony
06/12/87	Frank Defier	Deposition
06/15/87	Greenhill v. Dow	Deposition
06/16/87	Moss v. Charter Oak	Deposition
08/12/87	Kracht & Galletin	Deposition
08/12/87	Gilpatric Family	Deposition
08/13/87	Southard	Trial Testimony
08/18/87	Pikeville, Kentucky v. Monsanto	Deposition
08/24/87	Eileen Newblock	Deposition
08/26/87	Scott et al. v. Monsanto	Trial Testimony
09/16/87	Barnett et al. v. Hurst Graphics	Trial Testimony
10/07/87	Larry Unger & Clarence Pettit	Deposition
10/08/87	Virgil Dotty	Deposition
10/09/87	Santiago v. Firestone	Deposition
10/12/87	CAPCO Plaintiffs	Deposition
10/14/87	Eileen Newblock	Trial Testimony
10/16/87	Terry Bronder	Deposition
10/19/87	Lewis Gazaw	Deposition
10/20/87	Charles Corder	Deposition
10/21/87	Lloyd H. Sweger	Deposition
11/12/87	Clarence Pettit v. Armstrong World	Deposition
11/30/87	Greenhill v. Dow	Trial Testimony
12/2/87	USA v. Protex	Trial Testimony
	CALENDAR YEAR 1988	
01/15/88	Gazaw v. Air Products et al.	Deposition
01/16/88	Comeaux v. Sabine Propellor	Deposition
02/01/88	Santiago et al. v. Firestone et al.	Trial Testimony
02/27/88	Lloyd H. Sweger	Deposition
04/22/88	De La Garza v. Ashland Chemical	Deposition

DATE	CASE	TYPE OF APPEARANCE
04/23/88	Griffen v. Engineering Professional	Trial Testimony
04/24/88	Cook v. Armstrong	Trial Testimony
04/25/88	Jacobs v. Union Carbide	Deposition
04/26/88	Edmon Benavides	Deposition
04/26/88	James L. Nations	Deposition
07/07/88	Madra Weaver	Deposition
07/12/88	Skeen v. Monsanto	Deposition
09/15/88	Williams v. Memorial Hospital	Deposition
09/15/88	Santiago v. Firestone	Deposition
09/27/88	Miller v. Amax	Deposition
10/11/88	Evanson v. Osmose, et al.	Deposition
10/14/88	Wyels et al v. Neoplan	Deposition
10/26/88	Karos v. Vertac	Deposition
10/31/88	Santiago v. Firestone	Trial Testimony
11/14/88	Arvel Day v. Union Carbide	Trial Testimony
11/15/88	Newsome v. Texas City Refining	Deposition
11/29/88	Moss v. Charter Oaks	Deposition
12/05/88	Lockhart, Stricker, Kerr, Donohoe	Trial Testimony
12/09/88	Larry J. White	Deposition
	• • • •	

[Defendant's Exhibit No. 7-A]

LEGAL APPEARANCES BY DR. DANIEL T. TEITELBAUM

CALENDAR YEAR 1989

01/04/89	Alexander, Amon, Martinez	Deposition
01/20/89	Skeen v. Monsanto	Trial Testimony
01/30/89	John A. Martinez	Trial Testimony
01/31/89	Landrum v. Litton Systems	Deposition
02/08/89	Swartz v. Hewlett Packard	Mediation Testimony
02/15/89	Slaughter v. Farm & Home (Brio Site)	Deposition
03/03/89	Friel v. Paint Companies	Deposition
03/08/89	Stilley v. Katy SWD	Deposition
03/10/89	J.D. & Gladys Sanders v. Binford	Deposition
03/21/89	Dee & Charlene Byrnes	Trial Testimony
03/29/89	Nelson v. Farmland	Deposition
04/19/89	Cook v Fashion Carpets	Trial Testimony
04/26/89	Hynes v Armstrong World Industries	Trial Testimony
05/01/89	Askew v Red Devil Lye	Video Testimony
05/04/89	Kneppler v Alamo Barge	Deposition
05/09/89	Friel v Paint Companies	Trial Testimony

DATE	CASE	TYPE OF APPEARANCE
06/07/89	Nelson v Butler Paper	Deposition
06/22/89	Franco v Duraspan	Deposition
06/28/89	Fraley v Hospital Corp. of Am.	Deposition
07/11/89	Franco v Duraspan	Trial Testimony
07/20/89	Tice v Protex	Deposition
08/01/89	Howard Henderson	Video Testimony
08/03/89	Sipole v Union Carbide	Deposition
09/08/89	Hansen v Chevron	Deposition
09/21/89	Clarence J. McCall	Deposition
11/20/89	Hughes v Burlington Northern	Deposition
11/21/89	Porter v Union Oil CO	Deposition
11/27/89	Droddy v Texaco	Deposition
12/05/89	Brio Site	Trial Testimony
12/13/89	City of Laporte	Trial Testimony
	CALENDAR YEAR 1990	
01/04/90	Smith v Sabine Towing	Deposition
01/05/90	Fairley v Texaco	Deposition
01/08/90	Hansen v Chevron	Trial Testimony
01/12/90	Culp v Curtain Matheson, Inc.	Deposition
01/13/90	Cordova v Judwin	Deposition
01/16/90	Morrison v Eli Lilly	Deposition
01/22/90	Droddy v Texaco	Deposition
01/23/90	Baker v Armstrong Indus Inc.	Trial Testimony
01/24/90	Hudson v C F & I	[Deposition]
01/30/90	Quigg v Amoco	Deposition
02/06/90	Stone v Varn et al	Deposition
02/19/90	RE: Thomas Martin	Deposition
02/22/90	RE: Harold Washington	Trial Testimony
02/28/90	Wright v General Dynamics	Deposition
03/12/90	Harris v General Dynamics	Deposition
03/22/90	RE: Robert Hardy	Deposition
04/16/90	RE: ED Casey	Deposition
04/17/90	Zehr v Hoechst Celanese	Deposition
04/19/90	David F. Vaughn	Video Testimony
04/20/90	RE: ED Casey	Video Testimony
04/24/90	RE: Sorrells	Deposition
05/22/90	Zehr v Hoechst Celanese	Trial Testimony
05/23/90	Kucharski & Remmick	Trial Testimony
05/25/90	Flores et al. v Judwin	Trial Testimony
06/01/90	Paula Peters v. G.C. Hanford	Deposition
07/09/90	Davis v Avondale	Deposition
07/10/90	Robert Jenkins	Deposition

DATE	CASE	TYPE OF APPEARANCE
07/11/90	Keister v Dow	Deposition
07/18/90	Renaud v Martin Marietta	Summary Judgment Testimony
07/23/90	Gail Crull	Deposition
07/24/90	Akin & O'Brien	Deposition
07/30/90	Judy Cooper v State Farm	Deposition
08/07/90	Ray Spurlock	Deposition
08/14/90	Hofer v Amoco	Deposition
08/21/90	Carol Davis v Avondale	Trial Testimony
08/23/90	Keister et al. v Hercules	Trial Testimony
09/12/90	Ackerson et al. v Dow	Deposition
09/14/90	Green et al. v Marathon Oil	Deposition
09/18/90	Linda Atchley	Trial Testimony
09/21/90	Powell v Chevron	Deposition
09/25/90	Sean Kostelecky v Dow	Deposition
09/26/90	Arevalo et al. v Tide	Deposition
09/27/90	Gary Evenson	Trial Testimony
10/08/90	Stringfellow	Deposition
10/09/90	Betty Mayberry	Deposition
10/10/90	Cathey v Exxon	Deposition
10/11/90	Mullens v Wyeth Labs	Deposition
10/12/90	Mundo v General Dynamics	Deposition
10/24/90	Newman v Stringfellow	Trial Testimony
10/26/90	Buchaj v Texaco	Deposition
11/07/90	Green v Marathon	Trial Testimony
11/15/90	Linda Atchley	Trial Testimony
11/16/90	Rocha v Insta Foam Products	Deposition
11/27/90	Clair Adams, et al. v Westinghouse Electric, et al.	Deposition
12/10/90	Dole v Interstate Lead. OSHA	Trial Testimony
12/12/90	Sean Kostelecky	Trial Testimony
12/20/90	Sec. of Labor v Asarco OSHA	Deposition
	CALENDAR YEAR 1991	
01/10/91	Daniel McKee	Deposition
01/25/91	Larkin v Dow Chemical	Deposition
02/04/91	Johnson v Halliburton	Deposition
02/15/91	Chargois v Shell Oil Co.	Deposition
02/20/91	Daigle, et. al. v Shell Oil	Deposition
02/22/91	Wilbur Smith v Allied Signal	Deposition
02/26/91	Bartlett v Browning	Deposition
02/28/91	Pargmann v SPTC	Deposition
03/06/91	Wing v Shell Oil Co.	Deposition
04/23/91	Kolster v Union Carbide	Deposition

DATE	CASE	TYPE OF APPEARANCE
04/25/91	Jones v El Paso Products Co.	Deposition
05/15/91	Newman v Stringfellow, et al.	Deposition
05/28/91	Schamper v Dow	Deposition
06/04/91	Schamper v Dow	Evidentiary Deposition
06/21/91	Gerald Sheets	Deposition
06/24/91	Dupriest v Demco, Inc.	Deposition
07/02/91	Wyatt-Jose v. Eli Lilly	Deposition
07/09/91	Harvey v. Chevron	Deposition
07/09/91	Wycoff, et al. v. Chevron	Deposition
07/15/91	Lancaster v. Orkin	Deposition
07/18/91	Eubanks v. Shell Oil Co.	Deposition
07/23/91	Quante v. W.R. Grace, et al.	Deposition
07/24/91	Benoit v. Dixie Carriers, Inc.	Deposition
08/28/91	Best v. Walgreen Laboratories	Deposition
09/19/91	King, et al. v. Ameron Inc., et al	Deposition
09/20/91	Ellis, et al. v. Oxy Oil, Inc.	Deposition
10/18/91	Eubanks v. Shell Oil	Deposition
11/21/91	Fontenot v. ARCO	Deposition
11/22/91	Wright v. Mobil Chemical	Deposition
12/05/91	Oliver v. Kaiser, et al	Deposition
	CALENDAR YEAR 1992	
01/14/92	Wm. White v B.P. Chemicals, et al	Deposition
01/15/92	Larkin, et al v Dow Chemical Co.	Trial Testimony
03/05/92	Siemion v. USX, et al.	Deposition
03/19/92	Hathaway v. Flanders	Deposition
05/11/92	Acosta, et al v. Monsanto, et al	Deposition
05/13/92	Lancaster, et al v. Orkin	Trial Testimony
05/14/92	Previtt, et al v. Howard Hanna Real Estate Services, Inc.	Video Trial Testimony
05/20/92	James Infinger v. Ashland, et al	Deposition
05/21/92	Ronford Styron v. Shell, et al	Deposition
05/27/92	Shult, et al. v. Champion International	Deposition
05/28/92	Robert Escamilla, et al v. ASARCO, Inc.	Class Action Certification Hearing
06/04/92	O'Connor v. Mobile Chemical Corp	Deposition
06/10/92	Moyer v. Dow, et al	Deposition
07/27/92	Donald Campbell	Trial Testimony
08/05/92	Phillip Wilson	Deposition
08/06/92	Williams v. Burgess	Deposition
08/07/92	Shults v. Champion	Deposition
08/10/92	Jack Firmature	Deposition
08/17/92	Lockheed Litigation	Deposition

DATE	CASE	TYPE OF APPEARANCE
09/11/92	Escamilla v. Asarco	Deposition
09/16/92	Shults v. Champion	Trial Testimony
10/01/92	Aiken v. Unocal	Deposition
10/05/92	Donald F. Allen, et al.	Deposition
10/12/92	Steve LeBlanc	Deposition
11/02/92	Adkins v. Ashland Oil	Deposition
11/16/92	Lockheed Litigation	Trial
11/21/92	Jenna Nomeland	Deposition
12/04/92	Steve Marley	Deposition
12/07/92	Aiken v. Unocal	Deposition
12/22/92	Kent Woods v. Goodrich	Deposition
12/29/92	Cutshaw v. Weyerhaeuser	Deposition
	CALENDAR YEAR 1993	
01/05/93	Joseph P. Fiorella	Deposition
01/16/93	Richard Hammond	Deposition
01/20/93	Newman v. Stringfellow	Deposition
01/21/93	Lockheed Litigation	Trial
01/22/93	Belma Wright	Trial
02/17/93	R. Escamilla v. ASARCO	Trial
02/18/93	Elmer Monk	Deposition
02/19/93	Douglas Hoskins v. Oxy Petrochemical	Deposition
02/20/93	William Williams v. Burgess Products	Deposition
03/17/93	Cutshaw v. Weyerhaeuser	Trial
04/13/93	William Stagner	Deposition
04/14/93	Tom Morris v. Burlington Northern R.R.	Deposition
04/15/93	Chester Elrod v. Groendyke Transport	Deposition
04/16/93	Ferrel Fautheree	Deposition
04/19/93	Newman v. Stringfellow	Trial
04/23/93	Skaggs v. Simpson Lumber	Deposition
04/28/93	Diagle v. Shell	Deposition
04/29/93	Jack Walker	Deposition
05/05/93	Robert Moyer v. Dow	Deposition
05/11/93	Wilson Simon	Deposition
05/13/93	William Lee v. Interstate Lead	Deposition
05/28/93	Douglas Hoskins v. Oxy Petrochemicals	Trial
06/15/93	Kingsmill Community Water Supply v. Hoechst Corporation	Deposition
06/24/93	Duke Johnson v. Hoechst Celanese	Deposition
07/15/93	Carnival Cruise Lines	Deposition
07/27/93	J.W. Bartlett v. Browning-Ferris Industries Chemical Services, Inc.	Deposition
09/18/93	Billy Bane Smith/John Crawford v. Hartford Accident and Indemnity Co.	Deposition
10/05/93	Roy Adams v. International Paper Co.	Deposition

[Defendant's Exhibit No. 7-B]

10/06/93 — Daniel Thau Teitelbaum, M.D., P.C.
SELECTED DATA — CLIENT TIME DIARY

Init	Date	Hours	Description	Rate	Fees	Cumulative Tax	Code	Loc	Seq	Job
CLIENT #		33	Warshauer, Michael J.							
MATTER #		2578	Joiner vs. Westinghouse, Monsanto & GE							
EJ	07/06/93	1.00	Create File	50.00	50.00	50.00	19	B	1	H730
JLH	07/06/93	0.30	Index & file case material	90.00	27.00	77.00	29	B	3	H730
JLH	07/06/93	0.50	Drafting document	90.00	45.00	122.00	12	B	4	H730
JLH	07/07/93	0.20	Drafting correspondence for case	90.00	18.00	140.00	5	B	5	H730
JLH	07/07/93	0.30	Preparation of engagement Letter	90.00	27.00	167.00	21	B	6	H730
JLH	07/08/93	0.20	Research	90.00	18.00	185.00	7	B	7	H730
DTT	07/14/93	4.00	Consultation—meet with Mr. Holland and Mr. and Mrs. Joiner.	625.00	2,500.00	2,685.00	15	B	2	H730
JLH	09/01/93	0.20	Phone converation/follow up re: case	90.00	18.00	2,703.00	34	B	10	H930
JLH	09/09/93	0.30	Review document/correspondence	90.00	27.00	2,730.00	14	B	11	H930
JLH	09/13/93	0.30	Scheduling	90.00	27.00	2,757.00	56	B	12	H930
DKS	09/14/93	0.30	Compiling Literature/material for case	50.00	15.00	2,772.00	23	B	9	H930
JM	09/14/93	2.00	Professional Research Asso.-depo summary	150.00	300.00	3,072.00	74	B	15	H930
JM	09/15/93	2.50	Professional Research Asso.-depo summary	150.00	375.00	3,447.00	74	B	16	H930
JM	09/17/93	4.50	Professional Research Asso.-depo summary	150.00	675.00	4,122.00	74	B	17	H930
JLH	09/20/93	0.40	Preparation of deposition Letter	90.00	36.00	4,158.00	24	B	13	H930
JLH	09/20/93	0.20	Scheduling	90.00	18.00	4,176.00	56	B	14	H930
EJ	09/27/93	1.30	Library maintenance	50.00	65.00	4,241.00	36	B	8	H930
TOTAL MATTER		18.50								
TOTAL		18.50				4,241.00				

IN THE UNITED STATES DISTRICT COURT
FOR THE NORTHERN DISTRICT OF GEORGIA
ATLANTA DIVISION

(Title Omitted in Printing)

The deposition of DR. LARRY ROBERTSON was taken before Jennifer K. Young, Court Reporter and Notary Public in and for the State of Kentucky at Large, at Hyatt Regency, 400 Vine Street, Washington Room, Lexington, Kentucky, on Friday, October 8, 1993, commencing at the approximate hour of 10:00 a.m. Said deposition was taken on behalf of the Defendant, Monsanto, by agreement, for purposes of discovery and all other purposes as permitted by the Federal Rules of Civil Procedure.

* * * *

[5] The witness, DR. LARRY ROBERTSON, after first being duly sworn, was examined and testified as follows:

* * * *

[7] Q4 Let's identify which two articles you have in front of you.

A I'd be happy to do that. Let's take the first one first. This is an article by Lucy Anderson and other authors entitled, "effects of Polychlorinated Biphenyls on Lung and Liver Tumors Initiated in Suckling Mice by N-Nitrosodimethylamine," published in the Journal of the National Cancer Institute, volume 71, July 1983, pages 157 through 163.

* * * *

Q5 And the next article, please.

[8] A The second article that I am looking at is also an article by Lucy Anderson and other co-workers entitled, "Effects of a Single Dose of Polychlorinated Biphenyls to

Infant Mice on N-Nitrosodimethylamine-initiated Lung and Liver Tumors," published in the International Journal of Cancer, volume 38, pages 109 to 116 in the year 1986.

* * * *

Q6 When were you first contacted by someone about Mr. Joiner's case?

A I believe the initial contact was initiated by Michael Warshauer approximately the spring of last year.

Q7 What did Mr. Warshauer tell you about Mr. Robert Joiner, if anything?

A Basically that this gentleman is suffering from lung carcinoma, was currently—or at that time was in very poor health. He had an occupational [9] history of exposure by virtue of the fact that he was employed in the electric power industry in the capacity of repairing transformers, had suffered exposure to halogenated aromatic hydrocarbons. I was also told that he had, that is Mr. Joiner, had a history of smoking, cigarette smoking.

Q8 Anything else?

A That was the gist, I believe, of the conversation.

Q9 Were you told what kind of lung cancer he had?

A I don't recall.

Q10 Have you seen his medical records?

A Yes, I have seen them.

* * * *

[12] Q22 Have you ever seen Mr. Joiner?

A No, I have not.

Q23 Have you ever been to the place where he worked?

A No, I have not.

Q24 Have you been informed as to what kind of fluids were in the transformers upon which Mr. Joiner was working?

A Through the attorney, Michael Warshauer.

Q25 What fluid do you understand to be in the—

A The fluids were various mineral oils containing varying concentrations of PCB.

Q26 Anything else, any pure PCB fluid in transformers?

A I have no knowledge of that.

[13] Q27 Now can you tell me what small cell lung cancer is?

A I'd like to say from the outset that it is not my intention to give statements regarding clinical aspects of this case. I am a research scientist. My knowledge includes the mechanism of toxicity of polyhalogenated aromatic hydrocarbons, specifically in animal models. I have most of my adult life worked in the research laboratory. I am a Ph.D. and not an M.D. I would very much like to limit my statements to non-clinical aspects of this case as relates to mechanisms of carcinogenicity in the context of the relevant scientific literature.

Q28 Can you tell me what small cell lung cancer is?

A Not histologically nor—no. I cannot tell you what it is.

Q29 Can you tell me how many different kinds of cancer one may find in the lung from various causes?

A There are a multiplicity of cancers arising from a number of different cell types, from a variety of different causes. That is general knowledge[. . . .]

* * * *

[14] Q30 . . . As far as you know, is there any credible evidence from a scientific probability point of view that PCBs initiate small cell carcinoma in the lung in humans?

A PCBs are generally not recognized as initiating agents but rather are widely accepted as promoting agents in a number of tissues.

Q31 So the answer to that question is no?

A The answer to the question as it was stated if PCBs have been shown clearly to be initiators of carcinogenesis is no. They are rather classified as promoting type carcinogens, not initiating type carcinogens.

Q32 Now I want to be perfectly clear about what I am asking you. I'm going to ask you the same question

about promotion. Is there any credible evidence as a scientific probability that PCBs promote, and I emphasis, small cell carcinoma in the lung in humans?

A I know of none in that context, no, in humans.

Q33 Do you know of any in animals? This is with respect to small cell carcinoma in the lung.

A I know of studies, and we have identified one of [15] them, in which PCBs promote DMN-initiated carcinoma in the lung of mice.

Q34 Is that article small cell carcinoma, no, is it?

A It's not identified as such, that's correct.

* * * *

Q35 [. . .] Along that line, what opinions do you expect to give in this case?

A On the basis of the articles which we will discuss in which the propensity of PCBs or the characteristic of PCBs to promote initiated carcinoma in the lung of mice on the one hand, and on the other hand, the very unusually young age of which this individual, Robert Joiner, developed [16] lung carcinoma. I expect, based upon of course what further information comes to me, that is I qualify my expectation on that basis, that my—my expert opinion will be that this gentleman developed lung carcinoma at a very young age because the onset of that disease was promoted, supported, accelerated by his exposure to halogenated aromatic hydrocarbons.

Q36 In the first place, what was the initiator of his lung cancer in your opinion, cigarette smoking?

A It's certainly a possibility. Cigarette smoke is known to have components which have—which are stronger initiators than they are promoters. That is one reason that individuals who develop lung cancer from cigarette smoking require almost a lifetime of cigarette smoking before they develop lung cancer. Years of exposure, decades of exposure to cause sufficient damage within the lung cells that cell division takes place fixing the damage that was initially incurred, that is the initiating capacity, requires a long period of promotion to

lead to the overt disease. Mr. Joiner had a relatively short time span between his smoking exposure and the onset of the disease, unusually short. Something must have accelerated [17] that process and that something was most likely the chemicals to which he was exposed in the work place.

* * * *

[18] Q42 When you gave me your opinion earlier about it being—I don't know what the words were you used but you said something about it being a promoter in his case because of his young age or something of that nature.

A The facts are that he developed as a young man, I believe, with 37 years a carcinoma of the lung, a type that is, so I've been told, often associated with cigarette smoke. That in itself indicates to me that some other factor may have played a role in accelerating this process.

Q43 As I understand it, you cannot point to anything in [19] the literature that says that small cell lung cancer has been produced in any animal model.

A No, I cannot at this time.

Q44 Is there any epidemiological evidence in humans that PCBs are promoters in small cell lung cancer?

A I'm not aware of that.

Q45 You're not aware of any?

A I'm not aware of any.

Q46 From your background as I understand it, your field is toxicology.

A That's correct.

Q47 Are you Board certified?

A No, I'm not.

* * * *

[20] Q49 And I believe you've earlier told me that you're not a medical doctor.

A I am not a medical doctor.

Q50 You will not give any medical opinions.

A Would not give any clinical opinions.

Q51 Any clinical medical opinions; is that correct?

A I would prefer if you just separated things into clinical and basic research.

Q52 Your field then is basic research—

A That's correct.

Q53 —in animals.

A In animals, that's correct.

Q54 You're not an immunologist?

A I am not a immunologist.

Q55 You're not an epidemiologist?

A I have experience with cohorts but I am not trained as an epidemiologist.

Q56 You're not a veterinarian.

A I'm not a veterinarian. Maybe I should qualify the statement about the epidemiology. I have, of [21] course, taken courses in epidemiology and have a master's degree in public health from the University of Michigan, which gives me some experience in looking at public health related phenomena, whether that's—public health by definition, of course, deals with a population as opposed to the individual.

Q57 Any other opinions that you're going to give about Mr. Joiner specifically?

A I would like to limit my testimony to what's in the scientific literature, primarily mechanisms, toxicity relating to polyhalogenic aromatic hydrocarbons and those aspects of this case.

Q58 When you do scientific research, do you usually subscribe to some sort of scientific method—methodology?

A Of course.

Q59 Would you please describe that in detail for me? Science often starts with a question or a hypothesis. Mechanisms are formulated for addressing this question or hypothesis, proving it or disproving it. Experiments are designed. Animals, in my case, are ordered. Treatments are administered. Animals are sacrificed. Measurements are made. The question or hypothesis [22] is accepted, rejected or the data is insufficient to do either of those two things.

Q60 Earlier you had, I think, two papers marked that you had brought with you. Both of these papers are offered in part by Lucy M. Anderson.

A That's correct.

Q61 And when she refers to—looking here at Deposition Exhibit No. 2, to suckling mice, are those baby mice? What does that mean?

A That's correct. Those are mice that are suckling. That is they are drinking milk from their dams, from their mothers.

Q62 She dosed these mice. What kind of dose did she give them?

A First of all, she treated the dams, the mothers with PCBs on their ninth—these were pregnant dams, on their 19th day of gestation. The suckling mice themselves were treated either on day four or on day 14 with a potent alkylating agent, a carcinogen, dimethylnitrosamine.

Q63 And these little suckling mice—

A They grew up, yeah. Then they were killed either at 28 weeks of age or they were killed at 18 months of age.

Q64 They had some production of tumors; is that [23] correct?

A That's correct.

Q65 Was she able to reproduce that in adult mice?

A That question is not very clear to me. This study which was done in 1983, to my knowledge, was not attempted to be reproduced. And this second paper does not purport to be an attempt to reproduce this study. The design of these two studies are different.

Q66 Did you understand the question because I understand that she was not able to make the leap from the suckling mice or baby mice to adult mice? That's eitther true or not.

A Or the other alternative is I don't know.

Q67 Is that your answer?

A I don't know at this time. I do not—I cannot remember the specific paper in which exactly this experiment was attempted in adult mice. That paper may exist and I'll be happy to look at it if you can produce it.

* * * *

[25] Q72 First of all, do you know of any human epidemiological studies that relate to small cell lung cancer?

A Epidemiological studies don't usually relate to a single end point. Usually you look at a cohort for a variety of symptoms or signs that may occur as a result of an experience like an exposure to a toxic chemical.

Q73 Do you know of any that relate in humans to lung cancer in general that would illustrate that lung cancer is promoted by or encouraged or whatever by being exposed to PCBs?

A I am not aware of that exact kind of study. I think such studies have been attempted but I do not think they are published. They may be in fact ongoing. I think they would be very interesting to the scientific community to see those. I do know of an epidemiologic study where the joint function [26] of asbestos and cigarette smoke was clearly demonstrated.

Q74 Do you know about any—the answer about PCBs then is no?

A I'm not aware of such studies. I hope such studies will be done. I would look forward to reading such studies.

Q75 Now in animal work and specifically in the two papers that we had marked earlier, two and three I believe are the numbers, were the tumors produced in relation to the dose given?

A Yes.

Q76 How high a dose was given? Let's take Exhibit 2 first and then Exhibit 3. What were the range of dosage I should say?

A In the Exhibit 2, there was a—appears to be a single dose of 500 milligrams per kilogram whereas in Exhibit 3, there were a range of doses up to 500. That included a dose of 50, 250 and 500 milligrams per kilo-

gram of Aroclor 1254, I believe in both cases, yes. And I believe these were also single administrations in all cases.

Q77 When you say single administration, one dose?

A Yes.

Q78 How was the dose administered?

[27] A From a toxicologic point of view, that's not an exact definition. In this case it's much better to mention the applied amount. Call it a dose, that's fine. How were they administered? In the—

Q79 Given in the feet or intravenously or what?

A In Exhibit 2 the PCB was administered to the dam, the pregnant dam on the 19th and it was administered IP, and that means intraperitoneally. The needle is actually inserted into the peritoneum, being careful to avoid touching the uterus, as it was described in this paper, at right angles to the mid-line of the body. So into the peritoneal cavity, in solution, in an oil, into the peritoneum.

Q80 So the Jury will understand, what is the peritoneum?

A It is the body cavity that includes the mesentery, many of the vital organs excluding, of course, the heart and the lung, which is in the thorax. So basically a body cavity including most of the vital organs of the—

Q81 Stomach and the liver.

A The stomach, the intestines, exactly. In answer to your question, the second paper, I believe, they were also administered—no, excuse me. In Exhibit [28] 3 the doses of Aroclor were administered intra—by an intragastric dose, which in toxicology parlance means by gavage, which means basically a needle was put down the throat of the mouse and the material was introduced directly into the stomach, inside the stomach as opposed to the peritoneum which was outside the stomach.

* * * *

[29] Q85 [. . .] Using those terms, can you tell me what the level of exposure was to Mr. Joiner and what dose he got from any evidence anywhere in this case?

A No. I think his exposure to this class of compounds will have to be reconstructed on the basis of his work jobs, the activities that he was involved in on a daily basis, the levels of PCBs in those oils that were drained from transformers, his contact with those, and so forth. If I were asked to do that, what I would try to do is to go back and define tasks and define the time periods with which he was involved in those tasks. I would attempt to learn the levels of halogenated aromatic hydrocarbons in those oils with which he was in direct contact. And very importantly, I would want to know when fires occurred, critically important, when materials were heated. He was a welder I understand. He welded and repaired at times so he [30] brought high temperature in contact with surfaces that were potentially contaminated with PCBs and oils. So of critical importance is on which occasions accidents occurred, during which times high temperatures were applied to "bake out" coils that had been removed, literally dripping with mineral oils and PCBs from these transformers, what sort of ventilation there was in the work space, what sort of protective clothing this gentleman wore, when he was instructed to wear protective clothing, how this protective clothing was disposed of, whether or not he himself and his skin was directly in contact with these liquids. I think a careful analysis of these parameters could lead to a reasonable estimation of his exposure over those years. That would require very careful reconstruction and knowledge of situations.

Q86 Have you been asked to do that?

A No, I have not.

* * * *

[31] Q89 That's a different question than what I asked you. Do you have any information—

A I have heard that—that fires—

Q90 —that he was present when a fire occurred and if so, what is the source of that information?

A Since he repaired transformers, I assumed that he repaired transformers that had been struck, for example,

by lightning. So I would assume that he, until proven otherwise, that he was exposed to by-products of burning and very high temperatures associated with transformer fires.

Q91 Now for him to get any dose from that, what concentrations of PCBs would necessarily be present in the mineral oil, in your opinion?

A Dose is not dependent on the concentration of the PCB in the mineral oil. If there is PCB in the mineral oil, it has the potential to—for him to be exposed to it.

* * * *

[37] Q112 Earlier you testified that you understood that the mineral oil had varying degrees of contamination with PCBs.

[38] A That's correct.

Q113 Can you tell me what the variance was of what—

A No. I don't know what the levels were and I think you should establish that with other witnesses.

* * * *

[Deposition Exhibit 7]

TRIANGLE LABORATORIES OF RTP, INC.
POLYCHLORINATED BIPHENYLS ANALYSIS

File Name: W022015
Concal: W022005
Analyst: VC
Sample Size: 1.00 g
Ical Date: 02/04/92
Spike File: SPPCBC1K
Client: DR. LOUBE
Sample ID: ROBERT JOINER

Analysis Date: 02/05/92
Sample Matrix: ADIPOSE
Sample Origin: n/a
TLI Sample ID: 52-135-1
Project Number: 19710R
Date Received: 12/19/91
Date Collected: / /
Client Project: n/a

Name	Conc (ppt)	[No.]	DL	Empc	Ratio	RT	Flags
2-Mo	ND		26.9				—
44-Di	ND		23.8				—
244-Tr	3870				1.05	21:56	—
2255-T	ND		46.4				—
3344-T	ND		38.4				—
22455-Pe	314				0.59	27:47	—
224455-Hx	56330				1.25	31:51	—
2234455-Hp	36470				1.04	36:18	—
22334455-Octa	9640				0.91	40:31	—
223344556-Nona	5430				0.76	42:13	—
Deca	3070				1.14	43:44	—
TOTAL MONO	ND		26.9				—
TOTAL DI	ND		23.8				—
TOTAL TRI	3870	1			1.05		—
TOTAL TETRA	11680	4			0.79		—
TOTAL PENTA	35410	9		35810	0.68		—
TOTAL HEXA	117440	14		117650	1.24		—
TOTAL HEPTA	84310	13			1.02		—
TOTAL OCTA	35950	5		36220	0.87		—
TOTAL NONA	11200	3			0.78		—

Reviewed By: ———— 2/12/92

PCBC-RPT 4.01, LARS 4.20

IN THE UNITED STATES DISTRICT COURT
FOR THE NORTHERN DISTRICT OF GEORGIA
ATLANTA DIVISION

(Title Omitted in Printing)

DECLARATION OF MARK HOMYK

Mark Homyk gives the following declaration in writing subscribed by him, under penalty of perjury, as follows:

1.

He is the Director of Engineering and Electrical Operations for the City of Thomasville Water & Light Department, more than 21 years of age, and competent to give this declaration which is based on his personal knowledge.

2.

Beginning in 1983, the City of Thomasville Water & Light Department began having its mineral oil transformer dielectric fluid in its transformers and voltage regulators tested by independent laboratories to determine whether or not the transformers or voltage regulators were contaminated with PCBs.

3.

The transformers and voltage regulators which were tested by PCBs did not include the entire inventory of transformers of the City of Thomasville Water & Light Department, and the transformers which were tested were, in large part, selected for testing based upon their manufacturer serial numbers which from experience tended to indicate transformers which were possibly contaminated with PCBs, although not always.

4.

To the best of the undersigned's knowledge, the City of Thomasville Water & Light Department never purchased or used any transformers or voltage regulators containing pure Pyranol, pure Inerteen, or similar dielectric fluid mixtures of PCBs.

5.

The computerized information of the City of Thomasville Water & Light Department of PCB test results for mineral oil filled electric equipment, a complete and current printout of which is attached as Exhibit A, is maintained by the undersigned, who is the custodian, and the information is kept in the course of the regularly conducted business of the City of Thomasville Water & Light Department and it was the regular practice of the undersigned to make and maintain the computerized information as a compilation of test results received by the undersigned from time-to-time (as indicated on Exhibit A) from various independent testing laboratories.

I declare under penalty of perjury that the foregoing is true and correct.

Executed on 16 November, 1993.

/s/ Mark Homyk
MARK HOMYK

IN THE UNITED STATES DISTRICT COURT
FOR THE NORTHERN DISTRICT OF GEORGIA
ATLANTA DIVISION

(Title Omitted in Printing)

Deposition of ROBERT K. JOINER, taken in the above styled case on the 9th day of June, 1993, in the County Commission Room of the Thomas County Courthouse, North Jackson Street, Thomasville, Georgia; commencing at approximately 9:10 a.m., before Judy B. Scott, Certified Court Reporter B-1487, Georgia.

[2] ROBERT K. JOINER,

having first been duly sworn to tell the truth, testified as follows:

* * * *

Q Would you tell us your full name, please.
A Robert Kevin Joiner.
Q Mr. Joiner, how old are you, sir?
A I'm 39.
Q And date of birth?
A 2/9/54.
Q February the 9th, 1954?
A Yes, sir.
Q And where were you born?
A At Thomasville, Archbold Hospital.
Q And have you lived in Thomasville your entire life?
A Yes.
Q Who are your parents?
A Bud and Edna Joiner; both deceased.
Q What was your father's full name?
A Thomas Hall Joiner.

Q And your mother's full name?
A Edna Ophelia Joiner.
Q And when did your father pass?
[3] A November 13th, 1967.
Q And what was the cause of his death?
A A heart attack.
Q What was his age at death?
A Sixty- one. [58]
Q And when did your mother pass?
A Oh, God; '80 or '81, June the 11th of '80 or '81. [6/9/82]
Q What was the cause of her death?
A The final cause was brain cancer.
Q When you say final cause, does that imply that there were some other causes?
A Well, that was what she died of, brain cancer.
Q Were there other diseases that contributed to her death?
A Initially, she had lung cancer; no treatment. The book on that, it normally spreads to your brain. [Had cranial radiation after her cancer had spread to her brain.]
Q And what was her age at death?
A Sixty-one or -two. [64]

* * * *

[4] Q Did your father have cancer, also—
A No.
Q —prior to his heart attack? Any other history of cancer from your grandparents, uncles, or aunts?
A None, either side. [Uncle James mother's brother.]
Q So, your mother was the first member of your family, to your knowledge—
A Right. [No, second.]

* * * *

[18] Q [. . .] And the other two uncles?
A James Bloodsworth.
Q And who?
A And there was Louie Bloodsworth, but he died before I was born.

Q He died at birth?
A No, sir; he was—I don't really know his age. He was a young man, though.
Q And what about James, when did he die?
A In '77, something like that.
Q And do you know the cause of death?
[19] A I'm not sure. [Lung cancer.]
Q And do you know—
A My—
Q Excuse me.
A —Uncle James may have died from cancer. I'm not sure.

* * * *

[20] Q Now, did your father smoke?
A Yes.
Q Did he smoke all the time you knew him?
A Yes.
Q How frequently did he smoke?
A I was young when he died. I—he just smoked.
Q How old were you at his death?
A Twelve.
Q You just knew him to be a smoker?
A Right.
Q How about your mother, did she smoke?
A Yes.
Q And how frequently? A pack a day? Two packs a day?
A I don't know. She just smoked.
Q Would you characterize her as a heavy smoker or moderate?
A Compared to what?
Q To other smokers. You know, like some people smoke ten cigarettes a day, and some people smoke four packs a day. I would say four packs a day is a heavy smoker.
A No, not a heavy smoker.
Q Two packs a day?
[21] A No.

Q A pack a day?
A I would be just guessing—
Q You don't know?
A —and I don't want to do that.
Q No, I'm not asking you to guess. Did she smoke all the time up until the time of her death, that you remember?
A Yes.
Q How old were you when you left her home?
A I'm going slow to get all my facts straight. Twenty-three.

* * * *

[23] Q Did your father smoke cigarettes, a pipe, a cigar or—
A Cigarettes.
Q Cigarettes. Do you know what brand he smoked?
A Pall Mall.
Q Now, what about you, are you a smoker?
A No.
Q Have you ever been a smoker?
A Yes.
Q When did you smoke?
A These dates, you know, you don't write them down on calendars when you start or anything like that. I smoked approximately eight years.
Q When did you start smoking?
A I'm not sure exactly the date. I'm just not sure.
Q Was it in high school?
A I may have played around with them a little bit in high school, but not serious. No; uh-uh (negative response).
Q When do you remember first starting to be a serious smoker?
A I'm not sure. After high school.
Q Do you remember when you quit?
A The fourth Friday in April, 1980 or 1981 [1982]

* * * *

[25] Q So, that would mean you started when you were 18 or 19?

A That's right.

Q What kind of cigarettes did you smoke?

A I smoked some Marlboro, I believe; some Vantage; Salem Lights. [Some Winston.]

* * * *

[29] Q Okay. When you were smoking cigarettes during that eight-year period, how many cigarettes would you smoke on an average day?

A That's hard to judge, but the last two or three years, I would make myself a note or a mental note of if I open a pack at 7:00 a.m., I could not open another pack until the following 7:00 a.m.

Q So, a pack a day the last two or three years?

A A pack every 24 hours; right.

Q What about those first five or six years?

A Probably less.

* * * *

[46] Q Tell us about each job you've had.

A Well, I had the same job all the way through, just more responsibility at it.

Q And a different title, I would assume?

A Yeah. From Apprentice—at that time, they called the next step a "B" Electrician.

[47] Q When did you become a "B" Electrician?

A I'd just have to get the City records to tell you. If I told you, it would be just a pure guess.

Q What was next?

A "A."

Q And you don't know when that happened either?

A Not for—to the year, no. I would be—and then Chief, then brick wall.

Q That's where you are now, Chief?

A (Nodded in the affirmative.)

Q And you've been Chief for how long?

A Seven or eight years, just guessing.

Q When you say brick wall, it means there's no further level for promotion?

A It's hard to say. It's hard; it would be hard.

Q There's no natural progression to go from Chief Electrician to any other position? You're at the top of your category?

A Not without an electrical degree from Tech or somewhere. [. . .]

* * * *

[48] Q Did there come a time when you were a supervisor of other employees of the City?

A I've been a supervisor, a working foreman is what they call me, for a long time.

* * * *

[49] Q Now, when did you take on the supervisory position of working foreman?

* * * *

Q Give me your best estimate. Has it been ten years?

A It's just a guess. Eight or ten.

* * * *

[61] Q [. . .] Could you describe for us your duties at the City of Thomasville, from the time you started [62] in '73 through today. You know, just in a brief narrative, tell us what you've done.

A Transformer repair.

Q You're starting—are you going to start in '73 and come forward?

A Yeah. Like I told you earlier, just more responsibilities were added; nothing changed. Transformer repairs, regulator repairs, substations.

Q Substation repairs?

A Yes. Any electrical work related at the City, repairing lights or stuff. Now, that stopped, but we did all of that, you know, office lights and stuff. Take care of the airport; the peak shaving plant I was talking to you earlier about; motor compressors, motors, motor starters, water softener motors, motor starters.

Q For the Water Department?

A Wells and well starters. Now, that's as-needed repairs; but, our main function was substation and transformer, regulators, and that, you know. Those are our main functions that we do.

* * * *

[65] Q Now, over this period of 20 years, what percentage of your time would you say that you spent working on the repair of transformers?

A And regulators?

Q Well, I want to ask about transformers first.

A Transformers and in the area where they were being worked on?

Q Yes, sir. No, just tell me the time spent working on transformers first.

A Okay. I guess 40 percent or 50 percent of the time anyway.

* * * *

[66] Q So, would you say you spent half your time or 50 to 60 percent of your time doing that work, 40 percent of your time working on these transformers?

A It's hard to say. Probably around fifty-fifty. [50-50 some yrs. then 60-40 some years depending on weather how much time was spent at water softener & gas plant.]

Q Now, of the 50 percent of the time you're working on the transformers, how much of that time would you be working on the changing of the fuel or the oil in the transformer?

[67] A Well, depending on the transformer conditions.

Q Just approximate time, the percentage of your time, though.

A Probably 30 percent; 30 percent of the time. [90% 90%] See, it has to be pumped down before you can see any components in the transformer, to see anything. To inspect it, it has to be pumped. Every transformer—we have to open the top on every transformer that comes in. And if you do any work, you've got to pump it down

to get to it to work. And if you want to untank it, you've got to pump it down to get to the bolts, the attachment bolts, to untank it.

* * * *

[82] Q Now, on the transformers that you have worked on in the shop over this past 20 years, explain to us how you would come into contact with what we call the oil in the transformer.

A By opening the top of the transformer, pumping the oil, doing the repair work.

* * * *

[84] Q In coming in contact with the oil in these transformers, how would you come into contact with it?

A Well, you know, the top won't come off by itself, and you've got to take that off. Then if you want to work on it, you've got to pump some oil out of it. And we'd pump it out with a hose, and then take whatever mechanism we need to take off to work on it. If we wanted to untank it, we untanked it and pumped the oil out.

Q When you had pumped the oil out, where would you put the oil?

A Depending on the situation. If it was a large transformer, clean 55 gallon drums. A small transformer that we thought would take a—would be out of service or be untanked for maybe an hour or two, we might pump it into an open container because it was going back in; but, you didn't want to leave it out a long period, for dielectric—it loses dielectric properties.

* * * *

[94] Q Did the performance of this work require you to [95] come in physical contact with this oil?

A Sure.

Q How would you come in contact with it?

A Now, performance of what part of the work?

Q Any part of it. Just tell us how you would come in—

A Well, you know, if you're working on a transformer all the other years without any protection, no gloves, nothing, you're going to have to pump it out and you've got oil all over you. You're going to have to get in there and untank it. If it's a 500, you just about crawl in it to get in it and unbolt the frame from the tank.

Q So, you get the oil all over you?

A All over you at times. And a hose rupture or spill or all manners of things. You're dropping a tool, and it'd splash up in your face and junk like that. Even the dielectric test, you know, you was there when it was arcing and a little boil of smoke would roll out, you know, when you was testing the oil. It's just you was around—you was just there all the time, really.

Q You said in the early years before they had protective gear?

A Yeah.

Q When did the City furnish you with protective gear?

A This is a guess. I'll have to go back; but, '85 to [96] '87, in that area. [not sure about the date]

Q What did they furnish you with?

A Tyvek suits, Tyvek overshoes, full face respirator and approved filters.

Q And what about gloves?

A And gloves; nitrile PCB gloves or oil gloves, either one.

Q Do you remember when that—why that happened, why all of a sudden you were furnished with this safety gear?

A The EPA started, you know, pushing it, mandating it, and all this.

Q Is that something that other utilities had been using for a long period of time?

A I have no idea.

Q So, all you know is that they said, "Well, here's this protective gear"?

A Right.

Q Were you told to wear it?

A Right.

Q Did you wear it?

A Yes.

Q Always?

A Always, if I knew—now, wait a minute. Let's start over on that. What do you mean by always?

Q Anytime you—

[97] A Anytime I open any transformer?

Q Right; so that you wouldn't come in contact with the oil.

A No.

Q Why would you not wear the gear?

A When I knew—after all the EPA about this PCB's and stuff, when I knew they was non-PCB.

Q How would you know that?

A The only way we'll take a top off a transformer is it—we've got a punch that we punch the side, and we use gloves to retrieve the oil in a bellow and put it in a test vial and ship it off to a certified independent lab for testing. [or if the nameplate on transformer stated no PCBs.]

Q And then it's determined that it is non-PCB?

A Zero parts, then I will not use—I don't use—I won't use it then; but, if it's contaminated or PCB, definitely we've got to have it on.

Q If it had one part per million, then you would wear your protective gear; is that—

A No, I didn't say that. PCB contaminated.

Q If it has more than 50 parts per million?

A Right; right.

Q And you would not—ever since 1985, you have made sure that you wore your protective gear on any transformer that was PCB contaminated?

[98] A That's right; if I knew anything about it, that's right.

Q Or any transformer that would be—

A Suspect.

Q —a PCB transformer?
A Right.

* * * *

[99] Q So, it would be fair to say that since 1985, you've not had any contact with any PCB contaminated oil?
A No, that wouldn't be fair. [Im not sure of date]
Q Why would that not be fair?
A You don't know every transformer, and it's just too many occasions we're around it. We don't—we may not know, and I couldn't say that.
Q But so far as you know, any time that you worked on a PCB contaminated transformer or what might be characterized as a PCB transformer, you wore the gloves, the respirator, the body—
A Whenever we were required to do it, whatever year that was. I'm not sure on the year.
Q Right; we'll have to determine that.
A Okay.
Q But since that time, anytime—
A We try to protect ourselves.
Q Right. So, would it be fair to say that if you have had any contact with a PCB contaminated oil, that that would have to be rare since you've been so careful to make sure that didn't happen?
[100] A Yeah, whenever the date started.

* * * *

[101] Q And you had no knowledge of any reason that that could cause a problem to your health, of any kind, until about 1985 or '87, whatever that date is that we've [102] identified?
A That's correct.
Q And then after that time, you did everything you could to avoid any contact with any PCB contaminated fluid?
A Right.
Q So, I want to talk about prior to and let's—I'm going to use the date '85. I'm not holding you to that—
A I understand.

Q —but that has to be verified. Prior to '85, you made no effort to protect yourself from the contact with this oil in any way?
A No, sir [, but I'm not sure of the date].
Q So, it would come in contact with your skin?
A Yes, sir.
Q Did you ever drink it?
A Drink it?
Q Taste it?
A Well, they might have been a little bit accidentally splash, you know, from tools or something and get in my mouth, and I'd try to get it out; but, as far as drinking, no.
Q Did you ever swallow any of it?
A I have swallowed a little bit of oil, yes.
Q Do you know when you did that?
A No.
[103] Q Have you ever gotten it on your cigarettes and smoked it?
A Now, I just don't know about that. I wouldn't think so.
Q Have you ever gotten it in your eyes?
A Yes.
Q And how has that happened?
A Splashing and then not having eye protection.
Q How many occasions has that happened?
A Several occasions.
Q And what would you do when it was in your eyes?
A Try to get it out the best I could, you know, with water and get it out.
Q Did it burn?
A It burned, and it's very irritating. It's not a burn like alcohol, but it's an irritating burn.
Q Did you every report any incident like that?
A No. I should have I just didn't know the severity of it.

* * * *

[107] Q When were you diagnosed with the lung cancer?

A September '91. [August]

* * * *

[113] Q Now, as I understand it, you had told Dr. Johnson that you had had extensive exposure to toxic fumes and substances containing PCB's. Did you tell him that?

A Yes.

Q And that you had had heavy fume inhalations on several occasions?

A Yes.

Q Some involving fires?

A Yes.

Q Tell us about that, what occasions and—

A Well, we had a couple of transformers and regulator fires. But explaining about the smoke inhalation and all, we bake, what we called "bake-out," transformers that tested low dielectric. We'd untank them and put them on a three-wheel skidder, in the same area we all worked in, and take however many lights it'd take, depending on the size transformer, 1000 watt old ballpark lights and sit them around on step ladders around it and heat it up and get it way above ambient temperature. Smoke would boil off of them and in the same area where we worked. We done that numerous times.

Q Was that smoke just water vapor?

A No.

Q What do you think that was?

[114] A Well, I know it was.

Q What do you know it—

A Oil.

Q You think the oil was boiling off from the stadium lights?

A No, not boiling off. It's just the temperature of the core and coil was so hot that it was smoking. Oil was evaporating or diminishing off of it that way. We'd try

to get—we were trying to get the dielectric strength back up on the transformer core and coils.

Q Was there any heat being applied to the core and coils—

A Yes.

Q —other than the stadium lights?

A No.

Q So, the only heat would be the lights from these stadium lights that would be around the core and the coils?

A Within a foot or two. Those are very hot.

Q But just old stadium lights that came out of an abandoned football field?

A Correct.

Q How many lights would you put around the—

A Depending on the size of the transformer; a minimum of two, up to five.

* * * *

[115] Q So, that's the type fumes you're talking about, the fumes that would be generated by stadium lights mounted on—

A Smoke, yes, and several transformer fires. The tops were blown off.

Q Well, tell me about the transformer fires.

A Well, some of them would blow off the pole. I [116] mean, the tops would blow off; and, we would receive these transformers to work on, all charred and smoking, and breathing that mess.

* * * *

Q How many times did you work on a transformer where the top had blown off?

A It's hard to say. Not an every day occurrence, no; but, it's hard to say.

Q But by the time you got this transformer, there wouldn't be any fumes coming out of it?

A Sometimes not and sometimes, depending on how far that they'd have to deliver it, how long it would take.

Q In other words, you're saying it would be so hot when you opened it that there would be fumes coming out of it?

A Sometimes.

Q How many times did that happen?

[117] A Hard to say; it's some.

Q Would it be so hot that the oil was boiling?

A No. When these tops ruptured and blew off, a lot of times there would be—the oil level would be below the core and coils, and it'd just be smoking when they'd bring it in. Now, that wouldn't happen on an every day occurrence, but it's happened.

Q What about on these fires that you talked about, transformer fires?

A Well, we had a couple of fires in the substations.

Q When were they?

A In the '70s.

Q Where did they happen?

A What do you—

Q What substation?

A Metcalf Avenue is one of them. That was where we had the most problem.

Q Where was the other one?

A That was the main one there.

Q Well, you said there were two.

A A couple of times it happened over there.

Q Oh, two fires at Metcalf Avenue. What caught on fire?

A A regulator, a voltage regulator.

Q So, how would that effect the transformer?

[118] A Well, one time the reactor core faulted, and it shorted and spewed oil and caught on fire.

Q Oil out of the transformer?

A The regulator, the voltage regulator, which was the same manufacturers.

Q What kind of oil is in the voltage regulator?

A The same.

Q Who manufactured that voltage regulator?

A G.E.

Q G.E.?

A Uh-huh (affirmative response).

Q And so you're saying there was some oil that came out of the voltage regulator?

A Uh-huh (affirmative response).

Q And what happened to it?

A Fire.

Q And did you inhale the smoke?

A Yes. We put it out.

Q What other men were out there?

A I was the only one that got real close. There were several others out there. Hogan was in on one of those, but he wasn't real close.

Q What about the other incident?

A We had a main winding—the main winding, off the main winder of the regulator, it's very, very close to the [119] tank initially, and it faulted the ground there and burnt a hole in the side of the tank about two feet off the ground. It's about an eight-foot tank. All that oil run out and it caught on fire.

Q Did you inhale the smoke?

A I did.

Q And whose equipment was that?

A The same.

Q General Electric?

A Yeah. I was required to repair it.

Q Are there any other incidents where you believe that you inhaled fumes from this transformer oil or even the voltage regulator oil?

A Well, the biggest problem, the biggest amount was in the shop baking them out, that smoke.

* * * *

[120] Q Now, as I understand it, your cancer is now in remission?

A It's not evident on an x-ray, on the CAT scan. This is what their words were, "It's so much intensive [121] scarring, we cannot see any obvious at this time."

Q Are you presently undergoing any treatment?
A Not at this time.

* * * *

Q And you're not presently being treated in any way?
A Medication.
Q What type of medication?
A Anxiety; anxiety.
Q But not for the cancer, but for the anxiety?
A. Correct.

* * * *

[129] Q Did you have pneumonia when you were a child?

A I had bronchial pneumonia. I don't remember exactly how old I was, exactly; probably between 10 and 12.

Q Was this—this doctor said you had extensive left pulmonary disease, a major component of which certainly represents post-inflammatory residual from some sort of previous pulmonary disease, such as, pneumonia possibly with emphysema.

[130] A I had pneumonia when I was between 10 and 12.

* * * *

Q In your work place, during these years that you worked on these transformers, how many people in that work area were smokers? You smoked for awhile. How about these other men?

A. Had some; the majority not. I had such a turnover rate.

Q Is that a pretty confined area where you're working?
A It was, yeah.

* * * *

[143] Q Did you ever use any of the oil out of the transformers to start a fire to keep the men warm?

A Unfortunately, several times.

Q How would you do that?

A Take a 20 gallon drum—we had a 20 gallon drum and put oil-dri or anything about eight inches in it and put rags and soak it and light it and burn it.

Q When did you do that? When did that practice occur?

A We done some of that at the airport when it was cold days.

Q When did you discontinue that practice?

A We didn't do it that many times, but we did it; and, I don't know when it—it wasn't from somebody saying stop. It was we just quit doing it.

Q Do you know when?

[144] A No, sir.

Q This happened in the '70s or—

A Oh, yes. It definitely—it happened—I wouldn't have lit that up and done that knowing—after I quit smoking, I wouldn't have done that.

Q In 1980 or '81?
A Correct.

Q And that was because you just didn't want to inhale any smoke?

A That's correct.

* * * *

[145] Q Now, you said in your Answers to Interrogatories that you smoked Winstons. You said earlier this morning you smoked Marlboro. Which was it?

A Well, it's—I thought about that. It was some of both, but I wasn't a regular on either one of them; but, that was—some of both.

Q So, Winstons, Marlboros, Vantage, and Salem Lights?
A Correct.

* * * *

[183] Q [. . .] Now, I want to make sure I understood [184] this. To your knowledge, did the Water and Light Department ever purchase any transformers that contained Askerel dielectric fluid; or, were they all mineral oil transformers, to your knowledge?

A Not to my knowledge, they didn't, not to my knowledge.

Q When you say not to your knowledge—

A Because I wasn't over purchasing.

Q When you say not to your knowledge, do you mean—it's sort of a double negative there. To your knowledge, they never purchased an Askerel transformer; is that correct?

A To my knowledge.

* * * *

[195] Q So, it's on a three-wheel skidder. Was the purpose of baking the core to get the moisture out of the core so that you'd get your dielectric up?

A Correct.

Q It wasn't to boil the mineral oil off; it was to get the water out, the moisture?

A Well, you couldn't boil it out, anyway. You'd have to dry—you'd have to get a temperature well above ambient to dry it out, though, or you'd be there for months. You have to really heat it up, in my working with it.

* * * *

[197] Q When those were being baked, what would you be doing during that time; just going about your other responsibilities?

A Yes, sir; in the shop, repairing those, repairing other tools. We repair all the tools and all. Ordering; I'm responsible for ordering all the electrical equipment. My desk was about 50 feet from where the bake-out was.

Q Would you have also, during that time, have been going to the—let's see here—airport or the other places that you had to go?

A That's possible.

Q Now, when the steam would rise off of these cores—

A It's smoke.

Q —or smoke would come off of those cores, did it ever burn your eyes?

A Yeah; it could be very irritating if you—you know, you had to just be careful.

Q I mean, did you try to stay away from it when it was being baked?

A Well, there wasn't a whole lot I could do, if I had to work in that shop. And what was the worse is leaving the lights on and letting the door down for the weekend and let them continue to boil until you got back and all that smoke [198] was everywhere, and we done that.

* * * *

[199] Q But when you weren't baking spares to keep your spare inventory up, then you said you might go a long time without having to bake one?

A You may go a couple of months maybe without baking one.

Q What's the longest you think you ever went between baking cores?

A I really don't—I couldn't say for sure and be accurate.

Q Do you have any recollection of how many that you ever baked consecutively, one right after the other; any recollection of that?

A No.

* * * *

[204] And I take it the purpose for drying the core is you want to be able to use it when you get through, put it back in the transformer?

[205] A Correct.

* * * *

[206] Q Now, you mentioned two fires of voltage regulators. Do you remember any other fires involving any transformers, either pad mounted or pole type?

A Yes; we had several pole mounted transformer fires.

Q Do you recall whether you were involved in extinguishing any of those?

A I was not involved in extinguishing those.

* * * *

[207] MR. COCHRAN: Let's get the Court Reporter just to mark that as Joiner Exhibit Number 1, please, ma'am.

(Whereupon, Court Reporter marked exhibit as Joiner Exhibit 1 for identification.)

Q Take a look, if you would, Mr. Joiner. That's a document that you were kind enough to produce for us in this lawsuit. Is that a report that was kept by the Water and Light Department on one fire, where you went to the scene after the fire?

A Correct.

Q And the fire itself was extinguished by the fire department, was it not?

A They were sent to the area, but they didn't put the fire out.

Q Because the rain did; is that right?

[208] A Correct.

Q This occured in what year?

A '88.

Q So, this would have been after you were wearing protective gear?

A It was a late evening call out. Sometimes we didn't even know what the problem was until we got there, and we did not have the protective gear on our truck, the vehicle. All of this was in the shop. This is most of the—most of this kind of PCB work was lined-up, scheduled work; and, these were emergency situations, and we did not have it on the truck in every case, no.

Q Do you know whether you had it for this particular incident?

A I did not.

Q But in this particular incident, the only dielectric fluid that caught fire only had 26 parts per million?

A Correct.

Q And by the time you got there, the fire was already out?

A It was still smoking terribly, and it—but the fire itself was out; correct.

Q This says, "Rubber boots, gloves, plastic suits, brooms were placed in the DOT drums." What were those?

[209] A That's after the clean-up the following day.

Q How long were you there the evening?

A About 45 minutes.

Q What did you do while you were there?

A Went into the station and made sure everything was—all of the fuses were pulled, checked the transformer status, and set the top back on the transformer. It was scalding hot.

* * * *

[210] Q Did Dr. Shecter physically examine you?

A When you say physically exam, put his hands on me and—

Q (Nodded in the affirmative.)

A No.

* * * *

Q Where did you meet with Dr. Shecter?

A Where did I—

[211] Where?

A Did I meet him?

Q Yes. In Atlanta, where?

A At my lawyer's office.

Q That'd be Mr. Warshauer; right?

A Yes, sir.

* * * *

[213] Q [. . .] How many times have you met with Dr. Shecter?

A One time.

Q Now, when you met with him, was anyone present other than Mr. Warshauer?

A I believe all the other lawyers here were there. I believe they were.

Q Was your wife there?
A Yes.
Q Anyone else?
A There was a—I don't know what her position was.
Q Somebody that worked for Mr. Warshauer?
A Correct.
Q Anybody else?
A No, sir.

• • • •

[214] Q Now, you mentioned that your Uncle James had died of some form of cancer, but you weren't sure what it was. Did you have a second uncle who also died of cancer?

A That—Uncle James died of cancer.

Q Right. Was there another one, other than the Uncle James?

A No.

Q And you don't know what form of cancer that was?

A I can find out. [lung cancer]

• • • •

[223] Q What was the primary site of your mother's cancer?

A Initially?

Q Yes, sir.

A Lung cancer.

• • • •

[226] Q Now, when you were seen by Dr. Shecter—let's turn to Dr. Shecter for a minute. Did he prescribe any treatment for you? I mean, was he going to treat you in any way?

A Not at this time.

Q Was there any suggestion that he might treat you in the future?

A No.

• • • •

[231] Q Do you know of any other utility that would take cores out and bake them by clamping old stadium lights on step ladders?

A I don't. I don't know of any that did. I'm not saying they didn't.

Q You've never talked to any other utility about doing that?

A No.

Q Do you know who came up with the idea to do that?

A No.

Q Do you know how long they had been doing that at [232] the Water and Light Department before you got here?

A No.

• • • •

[Exhibit Joiner #1]

10 JUL. 88

(ASSUMED PCB CONTAMINATED) SPILL REPORT
LOCATION: CITY OF THOMASVILLE PEAK SHAVING PLANT LOCATED ON PINETREE BLVD WEST OF THE OLD ALBANY RD

DETAILS

DURING THE LATE EVENING OF 27JUN88 LIGHTNING STRUCK THE ELECTRICAL SYSTEM FEEDING A PAD MOUNTED TRANSFORMER BANK CONSISTING OF 4 TRANSFORMERS. THE WIRES, TRANSFORMERS, ETC. SHORTED OUT CAUSING A SMALL FIRE ON THE CONCRETED PAD. THE TOP OF ONE OF THE TRANSFORMERS HAD COME LOOSE DUE TO THE BUILD UP OF INTERNAL PRESSURED FROM THE ELECTRICAL FAULT AND SOME OF THE OIL HAD BOILED OVER THE TOP OF THE TRANSFORMER AND SOME OF THIS OIL CAUGHT ON FIRE AND BURNED. PROBABLY BETWEEN 20 AND 30 GALS OF OIL WAS GONE FROM THE TRANSFORMER. THE FIRE DEPARTMENT WAS SENT TO THE AREA, BUT THE RAIN WAS FALLING AT SUCH A FAST RATE THAT IT EXTINGUISHED THE FIRE. ROBERT JOINER (CHIEF SUBSTATION ELECTRICIAN) WAS CALLED TO ACCESS THE DAMAGE AND DETERMINE THE PCB LEVELS OF FLUID WHICH HAD ESCAPED CONTAINMENT. THE DEXSIL SCREENING TEST WAS PERFORMED ON ALL 4 TRANSFORMERS WHICH WERE AT THIS LOCATION AND THE TEST INDICATED THAT 3 OF THEM MIGHT BE CONTAMINATED WITH PCBS. LAB SAMPLES WERE TAKEN AND SENT FOR QUICK VERIFICATION TO " PPM ".

ON 28JUN88 THE MAIN PORTION OF THE CLEANUP OPERATION BEGAN. ALL EXPOSED WIRE, BROKEN BUSHINGS ETC. WERE PLACED IN DOT OPEN HEAD DRUMS. WHEN THE MISC. DEBRIS WAS REMOVED FROM THE AREA. WIPE SAMPLES WERE TAKEN ON THE CONCRETED PAD AND SOIL SAMPLES FROM THE AREA AROUND THE PAD. THE PAD WAS THEN CLEANED AND THE SOIL AROUND THE PAD DUG UP AND PLACED IN DRUMS. AFTER CLEANUP SAMPLES WERE TAKEN THEN FROM THE SOIL AND PAD. RUBBER BOOTS, GLOVES, PLASTIC SUITS, BROOMS WERE PLACED IN THE DOT DRUMS. THE FOLLOWING IS A LIST OF INDIVIDUALS WHO PARTICIPATED IN THE CLEANUP.

ROBERT JOINER
RALPH HOGAN
DAVID FOGLE
RONNIE PRANCE
ROBERT BURGESS
JAMES ADCOCH
JOE BATTLE
TIM WEAVER
MARK HOMYK
JOHNNY EDWARDS
FLOYDZELL SAMPSON

THE FOLLOWING IS A LIST OF THE TRANSFOMERS WHICH WERE LOCATED AT PEAK SHAVING PLANT

TX NO	MFG.	SERIAL NO	KVA	DEXSIL TEST	OFFICIAL LAB TEST
**2812	GE	H292457P70AA	167	GT50	26 PPM
1576	GE	C64941857P	167	GT50	287 PPM
1578	GE	C649006569	167	GT50	292 PPM
2822	GE	K175365Y71AA	25	LT50	

ON THE 28JUN88 TOM BERRY ACTING WATER & LIGHT DEPT. SUPERINTENDENT NOTIFIED THE DNR (SCOTT BALES ERT DIV OFFICER) AND THE NATIONAL RESPONSE CTR (TROUSDELL) WHO ASSIGNED THE #8636 TO THE INCIDENT.

LATE FRIDAY EVENING (1JULY88), I WAS NOTIFIED BY " PPM " OF THE RESULTS OF THE PCB TEST.

ON THE 5JULY88 I NOTIFIED THE OFFICE OF THE ERT DIV OFFICER AND THE NATIONAL RESPONSE CTR OF THE LAB TEST.

** WAS THE ONLY TRANSFORMER FROM WHICH ANY MATERIAL SPILLED OR BURNED DURING THE ABOVE DESCRIBED INCIDENT.

MARK W. HOMYK
Distribution Engineer

[Deposition Exhibit 5]

AFFIDAVIT OF ROBERT JOINER

Personally appeared before me, the undersigned officer duly authorized to administer oaths, Robert Joiner, who after being duly sworn deposes as follows:

1.

I am Robert Joiner, a competent adult. I am 37 years old and was born on February 9, 1954. I make this Affidavit for any lawful use or purpose.

2.

I have personal knowledge of the facts set forth in this Affidavit.

3.

I have been an employee of the Thomasville Electrical Cooperative since the Summer of 1973. At that time my position was classified as Apprentice: Substation, Electrical. My duties included repairing, cleaning and maintaining electrical transformers, oil testing, regulator repairs, and line repair items. In 1975 or 1976, I was promoted to Foreman; a position I held until the onset of my disability in August of 1991. As Foreman, my duties were expanded to include those of electrical repairman for the airport and primary/SECC underground cable repairman.

4.

Throughout the course of my employment with Thomasville Electrical Operative I was routinely exposed to Polychlorinated Biphenyls or, PCBs. The nature of my work caused me to inhale PCB fumes and endure the absorption of PCBs through my skin. The PCBs were contained in dielectric oils used in electrical transformers manufactured by General Electric and Westinghouse Electric.

5.

I inhaled PCBs while performing repair and maintenance work on transformers, as well as during instances of fire.

A.) On occasion I was required to repair large transformers. Once the oil containing PCBs was be drained, I would enter the transformer to make the necessary repairs. The transformer's inside casing usually continued to drip oil during the repair process, and oil also accumulated at the base of the transformer. I normally placed cardboard beneath me to avoid having to lay or sit in, the oil while working. The area in which I worked was almost completely enclosed, with the only vent hole being approximately 18 inches wide. The work space was very confined and extremely hot. My tasks usually required several hours work, during which I continuously breathed oil fumes laden with PCBs.

B.) Smaller transformers were delivered to and repaired at, the shop. The repair work required me to drain the transformer cases of oil and then, in the enclosed shop, heat the transformer cases under heat lamps to dry them out. During this process, the vapor from the oil containing PCBs filled the shop, and I inhaled it for many hours at a time.

C.) I often repaired transformers that had varying degrees of insulation contamination or, break-down. In these instances, the transformer's coils were untanked and placed under (four) 1500-Watt field lights in order to remove moisture from the winding. During the drying process, the transformer core and coils became so hot as to emit large quantities of smoke and fumes. The transformers were "baked" for seven or eight day periods, during which I was forced to work in the smoke-filled shop and inhale the PCB fumes.

D.) The most serious cases of insulation contamination/breakdown resulted in fire or, explosion of the transformer, both of which caused me to inhale concentrated fumes containing PCBs.

E.) I also inhaled PCB laden fumes while doing repair work from atop the transformer through the hand hole cover. I recall a particular emergency repair on a secondary connection, wherein the transformer's extreme temperature forced me to lay on cardboard connection, wherein the transformer's extreme temperature forced me to lay on cardboard while working. The PCB containing oil was so hot it had to be pumped down, and the combined heat sources caused fumes to rise. Since I was positioned on top of the transformer, I was forced to breathe the PCB fumes for the duration of the repair work—approximately four hours.

6.

The absorption of PCBs through my skin occurred during repair tasks, oil testing, and the " "untanking" " of transformers.

A.) The repair of small transformers involved submerging my hands and forearms in the chemical liquid contained inside the transformer case. In order to manipulate windings, I had to extend my hand down through the hand hole into the case. Other repairs required removing the top of the transformer and pulling out the inner core; whereupon, the PCB containing oil was allowed to drip into a pan before the core was serviced. Both repair methods involved skin contact with the oil containing PCBs; and often times, this chemical liquid splattered onto my clothing.

B.) Due to valve malfunctions, I was occasionally sprayed with oil containing PCBs while collecting samples for oil testing. At times the oil samples themselves spilled onto my hands, clothing, or shoes during transport.

C.) Prior to 1983, I was involved in the " "untanking" " (drainage) of large transformers. This process

often lent itself to accidental spillage, gushing valves, and random dumping of the chemical liquid. Upon completion of the " "untanking" ", my clothes, hands, and shoes were often covered with oil containing PCBs.

7.

During the period 1973 through 1983, I was unaware of the harmful effects of PCBs. The manufacturers of the transformers did not advise me of precautionary handling methods, nor was protective clothing or equipment recommended to me. The transformers upon which I worked bore no warnings or hazard signs concernings PCBs.

8.

I was not exposed to PCBs outside of my employment with Thomasville Electrical Cooperative.

9.

In August of 1991, I was diagnosed as suffering from squamous cell carcinoma (a form of lung cancer). This disease resulted from my occupational exposure to PCBs.

/s/ Robert Joiner
ROBERT JOINER

Sworn to and subscribed before me this 26th day of December, 1991.

/s/ [Illegible]

Notary Public, Thomas County, Georgia
My Commission Expires August 20, 1994

UNITED STATES DISTRICT COURT
NORTHERN DISTRICT OF GEORGIA
ATLANTA DIVISION

(Title Omitted in Printing)

The deposition of DR. PHILIP COLE, taken pursuant to Notice and agreement for cross-examination, discovery and any other purpose allowed by law and pursuant to the Federal Rules of Civil Procedure and the Federal Rules of Evidence; all formalities waived, excluding the reading and signing of the deposition; before Joyce S. Oglesby, Certified Court Reporter and Notary Public in and for the State of Georgia; commencing at 12:10 p.m., Thursday, October 21, 1993 at 4000 One Peachtree Center, 303 Peachtree Street, Atlanta, Georgia.

* * * *

[5] DR. PHILIP COLE

was called as a witness herein and, having been first duly sworn, was examined and deposed as follows:

* * * *

[10] Q. This next document I'm handing you is Plaintiffs' Exhibit No. 4. It's captioned "Smoking and Small Cell Lung Cancer." What is this?

A. This is a document that was prepared by me in August of this year, and it relates to what I consider to be a few of the salient aspects of that disease, that is small cell lung cancer.

* * * *

[24] A. So, I understand the question to be, if I may rephrase it: Is it true that you now hold the position that PCBs could not be a cause or a contributing factor in this particular case?

Q. Correct.

A. Okay. My response to that is that I would not make a statement that was as absolute as that question calls for, but I would say that in my judgment it is virtually certain that it was not a cause in this case.

Q. Or a contributing factor?

A. Well, I thought we'd been over that and had [25] agreed that cause and contributing factor were synonymously referred to as cause.

* * * *

[46] Q. Dr. Cole, are you aware of any harmful effects of PCBs on human beings? And I'm talking for purposes of this question short of an acute dose that would cause injury within a very short period of time.

[47] A. I'm not real sure of this, but I think that under some circumstances they can cause dermatitis or an inflammatory reaction of the skin, but apart from that, I—I'm not aware of any adverse health effect.

Q. How about of polychlorinated dibenzofurans?

A. I—I don't know.

Q. How about of polychlorinated dibenzodioxins?

A. With regard to the furans, I guess in high dose they do cause acute—an acute syndrome, sometimes called Yusho disease, Yu-Cheng disease. I—I really don't know exactly what the components of that are. As far as the polychlorinated dibenzodioxins are concerned, some of them, at least one of them, is—is quite a potent poison.

Q. With respect to the furans, you mentioned that you're aware of the acute effects, which you've described as—I guess referring to the Yusho or rice oil poisoning. And the longer term, more chronic effects, are you aware of any of those?

A. No.

Q. And of the PCBs, except for the dermatitis which you described, are you aware of any long-term problems that have been associated with PCBs?

A. No, I'm not.

Q. And with respect to the dioxins, you [48] mentioned that at least one of the congeners of dioxin is poisonous or acutely poisonous. I can't remember your exact word.

A. Well, —

Q. Are you aware of any long-term problems associated with dioxins?

A. Long term in the sense that it persists, but short term in the sense that it has a relatively short induction period with the chloracne.

* * * *

[49] A. I only meant to say that with regard to the question, "Are there diseases caused by these classes of chemicals and which make their clinical appearance after a long induction period?" my answer was, "I do not have a response to that. I do not know."

Q. Let me make sure that I've covered the waterfront on these chemicals. Are you aware of any disease processes that may begin shortly or immediately [50] following exposure but have a long life themselves?

A. The answer to this question is yes. Those conditions are chloracne and some of the signs and symptoms of the disease complex known as Yusho or Yu-Cheng, and those are the—those are the only ones that I'm aware of.

* * * *

A. And the question is: Is there any cancer in human beings that is known to be caused or promoted by PCBs?

Q. Yes.

A. No.

Q. How about dioxins, the same question?

A. My position is that no human cancer is caused or promoted by dioxins, but I do understand that this is an area of some controversy.

[51] Q. By your use of the last phrase "this is an area of some controversy" in your response to the dioxin question and your failure to use a similar phrase in response

to the question concerning PCBs, am I to assume that it's your position that there is no controversy concerning carcinogenic properties of PCBs?

A. With the understanding that one or two people won't make a controversy, the answer would be yes.

Q. Do you agree that there are at least some scientists who appear to be adequately trained who believe that PCBs do contribute to certain kinds of cancers in humans, sir?

A. I don't doubt that there is such a person or some number of people, but I couldn't name one for you. I really don't—I don't know of any.

Q. Looking at your Curriculum Vitae, in the teaching aspect of it, page 3, at the University of Alabama in Birmingham, is that where you presently are employed?

A. Yes.

Q. It says that you have taught epidemiology of cancer there?

A. Yes.

Q. The principles of epidemiology research?

A. Correct.

[52] Q. Advanced epidemiologic methods?

A. Correct.

Q. And that you've put on a doctoral seminar?

A. Correct.

Q. And those are also in the epidemiologic field?

A. Yes.

Q. Do you train people in your program to follow the correct methods of epidemiological science?

A. Of course.

* * * *

[56] Q. How many people does it take or what percentage of the scientific community in a particular field of study must hold the opinion for it to be recognized?

A. I can't—I can't answer that, except to point out to you that generally scientists don't divide themselves 50/50 on a—on a question, except in the earliest phases of it, that it then moves to 95/5 or 5/95. So, as long

as we're in the area where people are not taking positions, then it's a cause/effect relationship that is, in my parlance, not recognized.

Q. The bottom line of our last few questions, I guess, other than those concerning methodology or definitions of words, I want to make sure I understand, it's you opinion from your readings that there are no cancers in humans recognized as having been caused by exposure to PCBs?

A. That is correct.

Q. But do you agree that there are at least some scientists who hold the opinion that there is a correlation between PCBs and certain cancers?

A. Now, you've not only changed—

[57] Q. Oh, I have changed—

A. —the question from my view to what I think scientists, but you've also changed cause to correlation.

Q. Okay. Well, stay with cause, and then we'll go to correlation.

A. The question is: Do I recognize that there are credible scientists who hold the view that PCBs are an established human carcinogen?

Q. Yes.

A. The answer is that I cannot deny that there are such people, but I don't know who they are. I can't name anyone for you.

* * * *

[74] Q. We talked about benzene as a chemical of which you're aware is a human carcinogen for certain organs. Are you aware of any other chemicals which you recognize as human carcinogens?

A. Well, firstly, I did not acknowledge benzene to be a human carcinogen for certain organs.

Q. Okay. You gave me a disease, and I apologize for misstating that.

A. Okay. Now, you're asking me am I aware of any other chemical which is a recognized human carcinogen?

Q. Yes, sir.
A. Okay. The answer is yes.
Q. What are those?
A. There is a list of these compounds that I would be pretty willing to subscribe to, but I'll—I'll answer your question from my memory.

We've already mentioned benzene, Diethylstilbestrol, beta naphthylamine, 4-aminopbiphenyl, aflatoxin, bischloromethyl ether.

* * * *

[75] Q. Where would you—
A. Was there a favorite one of yours that you'd like me to agree to?
Q. You mentioned that there's a list. Where would I go to find a list that you would recognize? I know you just work from memory, and you may have inadvertently included one that's not on the list or included one—failed to include one that is on the list. I accept that as a possibility. Where would I go to find a list that you would recognize?
A. First, let me tell you that I didn't mention one that would not be on the list.
Q. Okay.
A. But I certainly did fail to mention some that—that are on the list.

The list that I like best, which is not to say that I subscribe to it across the board, is the list of Class I carcinogens put forward by the International Agency for Research on Cancer.
Q. IARC?
A. Correct.
Q. Do you recognize that group as a group that [76] is—whose opinions on cancer should be given a certain amount of credibility?
A. Let me say that there is no such thing as that group. The IARC is, in the context of which we're speaking, a convening body. It is not a authoritative body in and of itself. So, if I change your—if I change your question to be, "Are the results of the workshop

groups convened by the IARC generally to be accredited?" the answer's yes, generally.
Q. Can you think of specific examples which you do not give credit to?
A. Sure, I'll be glad to give you one.
Q. Okay.
A. They—This does not pertain however to Class I carcinogens, but to Class II. They consider, as an example, formaldehyde to be a Class II-A carcinogen. I would put it as a Class II-B. The same could be said for butadine. Those are two examples.

* * * *

[78] Q. No, I don't, but I would like to know how you believe those principles are applicable to your opinions that PCBs didn't have anything to do with Mr. Joiner's lung cancer.
A. Let me give you the short answer and see if that suffices. I think that that statement was intended to imply that there is a body of epidemiologic information that speaks to that question. That body of information includes about eight or so studies, that each of those studies can be evaluated individually, that the body of information that they comprise can be evaluated collectively, and that from those evaluations in the context of a set of criteria for making interpretations from individual studies and for making an interpretation from a body of knowledge collectively, one can deduce that PCBs have been shown not to cause cancer of the lung in human beings and that, therefore, they could not have caused Mr. Joiner's disease and furthermore, from the larger set of criteria that Mr. Joiner had an alternative cause of lung cancer which he experienced and that as a result of those two propositions, it is a virtual certainty that Mr. Joiner's disease was caused by his [79] cigarette smoking and that his occupational exposures contributed nothing to this.

* * * *

[87] Q. Well, from the studies you've read and your research into this area, do you think or do you believe

that there's any need to do any more research in the area or has it been established to your satisfaction as a scientist that these things are never going to be shown to be human carcinogens?

A. The context of your question of whether or not any more research is needed is from the specific point of view of showing them conclusively to be or not to be human carcinogens?

Q. Yeah.

A. Mr. Warshauer, I don't want to speak to that because conclusiveness in the sense in which would mean that almost everybody holds only one point of view is something a little difficult to achieve.

Q. Well, it's been achieved in cigarette smoking?

A. Yes, it has.

Q. Has it been achieved in benzene?

A. Well, now, wait a minute. You're talking about carcinogens. Here, we're talking about something that is virtually certainly not a carcinogen. So, the—the burden of proof for showing the absence of an effect [88] is substantially greater than that for showing no effect when you speak about the broad scientific community. So, if you ask me the question do I believe that the evidence is—is clear on PCBs and human carcinogenesis, I can't answer that, but what it will take to whip everyone into line, I don't—I don't know.

Q. In other words, people who are still studying it have reason to continue to study it because there are indeed people who aren't, quote, whipped into line?

A. Mr. Warshauer, I don't know why someone would choose to do yet another study of PCB carcinogenesis. All I can tell you is that I would not. I think it so unlikely to be rewarding that I don't want to spend a significant portion of the remainder of my career running up that alley. Here, you have a body of evidence that doesn't give the slightest inkling that these things cause human cancer. If—Surely if they were human carcinogens, we would see some evidence of it here (indicating), and we don't.

* * * *

[113] Q. Does one's risk of lung cancer diminish after one ceases smoking cigarettes?

A. The answer to that is probably yes, but that's not quite so clear in the form of lung cancer that he had, and also in any case probably doesn't happen to any appreciable extent for 10 or so years subsequent to cessation.

Q. Does it happen to an appreciable degree in seven or eight years?

A. Probably not, especially for small cell.

Q. What is unique about the small cell aspect of this case?

A. The literature on small cell suggests that the reduction in risk subsequent to cessation occurs more slowly, but admittedly, this is not a firm conclusion.

* * * *

[114] Q. Now, Mr. Warshauer also identified Plaintiffs' Exhibit No. 4, which is entitled "Smoking and Small Cell Lung Cancer," and I notice that on the first page of this, it has a heading "Association with smoking," and then it talks about, and let me just quote [115] this. It says, "SCLC," which I assume is an abbreviation for small cell lung cancer, "is strongly associated with smoking. Estimates of the relative risk," and then in parentheses it has "odds ratio varies from 5.0," a dash, "42.5 for current smokers."

What does that mean?

A. This means that the literature says that current smokers, and by implication, really ever smokers, have a risk of—Indeed SCLC stands for small cell lung cancer. —that is increased anywhere from five fold to about forty fold or the SMR terms that we were using before is anywhere from five hundred to four thousand or two hundred or so. In other words, there's a massive association between SCLC and cigarette smoking.

* * * *

[Plaintiff's Exhibit 1]

CURRICULUM VITAE

Born: August 22, 1938
Boston MA

Name:

Philip Cole

Position:

Professor and Chairman
Department of Epidemiology
School of Public Health
-and-
Associate Director for Epidemiology
Comprehensive Cancer Center
University of Alabama at Birmingham

Address:

Department of Epidemiology
School of Public Health
Room 203, Tidwell Hall
UAB Station
Birmingham, Alabama 35294

Telephone:

(205) 934-6707

Fax:

(205) 934-8665

Education:

Michigan State University	B.A.	1960
University of Vermont	M.D.	1965
Harvard University	M.P.H.	1967
Harvard University	Dr.P.H.	1970

Previous positions:

Department of Epidemiology Harvard School of Public Health	
Assistant and Associate Professor	1969-78
Professor	1978-79

Consultant in Epidemiology and Biostatistics Unit of Epidemiology International Agency for Research on Cancer	1977-78
Surgical Intern, Royal Victoria Montreal Hospital	1965-66

Certification and Professional Societies:

Licensed, Alabama Medical Licensure Commission	1981
Member, American Epidemiologic Society	1973-79
Certified, American Board of Preventive Medicine	1971
Licensed, Board of Registration in Medicine, Commonwealth of Massachusetts	1966-1980
Diplomate, National Board of Medical Examiners	1966

Honors:

American Cancer Society, Faculty Research Award	1973-78
Merk Lecturer, Montreal Cancer Institute	1977
Gordon Richards Memorial Lecturer, Ontario Cancer Treatment and Research Foundation	1979
John Whittick Memorial Lecturer Canadian Cancer Society	1980
Kammer Merit in Authorship Award American Occupational Medical Association	1982

John Rankin Visiting Professor of Occupational and Preventive Medicine
University of Wisconsin, Madison 1983

Eleanor Leader Memorial Lecturer
University of Toronto, Toronto 1985

Grand Prix Lacassagne du La Ligue Nationale Francaise contre le Cancer (with B. MacMahon, J. Brown and D. Trichopoulos) 1986

First Annual President's Award Outstanding Teacher
University of Alabama at Birmingham 1991

Major Committees:

Scientific Advisory Committee
Division of Cancer Cause and Prevention
National Cancer Institute 1978-80

Epidemiology and Disease Control Study Section
National Institutes of Health 1973-77

Clinical and Epidemiological Research Advisory Group
National Cancer Institute of Canada 1973

Committee on Epidemiology and Prevention (Chairman)
National Bladder Cancer Project
National Cancer Institute 1971-73

General Motors-United Auto Workers Occupational Health Advisory Board 1982-87

Mott Prize Selection Committee
General Motors Cancer Research Foundation 1985

Prevention, Cancer Control (Chairman) Steering Committee for the National Planning Effort
National Cancer Institute 1984-85

Board of Scientific Counselors
Division of Cancer Prevention and Control
National Cancer Institute 1986-90

Scientific Advisory Committee
Pittsburgh Cancer Institute 1987-89

Advisory Council on Epidemiology
Electric Power Research Institute 1986-90

Teaching:

Harvard School of Public Health
The epidemiology of chronic diseases 1969-72
The epidemiology of neoplastic diseases 1973-77
Epidemiologic methods 1976
Principles of epidemiology 1978-79

University of Minnesota—Graduate Summer Session
The epidemiology of cancer 1971, 74-80
Principles of epidemiologic research 1985
Fundamentals of epidemiology 1986, 87

International Agency for Research on Cancer
Cancer epidemiology 1974, 76, 78, 80

University of Massachusetts-Graduate Summer Session
Principles of epidemiology 1981-84
Cancer epidemiology 1982

Tufts University—Graduate Summer Session
Epidemiologic bases of public health policy and law 1986, 87

University of Alabama at Birmingham
Epidemiology of cancer 1980

Principles of epidemiologic research 1980-91

Advanced epidemiologic methods 1981

Doctoral seminar 1981-91

University of Michigan—
Graduate Summer Session
Principles of epidemiology 1989-91

Research Interests:

The epidemiology of breast cancer,
Hodgkin's disease and bladder cancer
Health effects of exogenous hormones
Occupational and chemical carcinogenesis
Health effects of electromagnetic fields

Editorships:

Associate Editor, Cancer Research 1982-85

Associate Editor, American Journal
of Epidemiology 1982-88

Editorial Board, International Journal of
Breast and Mammary Pathology 1984-

Editorial Board, Fundamental and
Applied Toxicology 1984-

Editorial Board, Southern Medical Journal 1990-

Publications: See attached list.

Prepared: January 2, 1992

Publications

1. Cole P, MacMahon B, Aisenberg A: Mortality from Hodgkin's disease in the United States: evidence for the multiple-aetiology hypothesis. *Lancet* 2:1371-1376, 1968.
2. Cole P, Gutelius J: Paraplegia resulting from the use of the subclavian artery as a shunt source during resection of the descending thoracic aorta. *Ann Surg* 169:293-294, 1969.
3. Rapoport A, Cole P, Mason J: Correlates of survival after initiation of chemotherapy in 142 cases of Hodgkin's disease. *Cancer* 24:377-381, 1969.
4. Cole P, MacMahon B: OEstrogen fractions during early reproductive life in the aetiology of breast cancer. *Lancet* 1:604-606, 1969.
5. MacMahon B, Cole P: Endocrinology and epidemiology of breast cancer. *Cancer* 24:1146-1150, 1969.
6. Cole P, Gutelius J: Neurologic complications of surgery on the descending thoracic aorta. *Can J Surg* 12:435-443, 1969.
7. Kaplan S, Cole P: Factors affecting response to postal questionnaires. *Br J Prev Soc Med* 24:245-247, 1970.
8. MacMahon B, Cole P, Lin TM, et al.: Age at first birth and breast cancer risk. *Bull WHO* 43:209-221, 1970.
9. Cole P, Monson RR, Haning H, Friedel GH: Smoking and cancer of the lower urinary tract. *N Engl J Med* 284:129-134, 1971.
10. Mirra A, Cole P, MacMahon B: Breast cancer in an area of high parity: Sao Paulo, Brazil. *Cancer Res* 31:77-83, 1971. Reprinted in Portuguese in *Rev Assoc Med Bras* 18:357-364, 1972.

11. Cole P: Coffee-drinking and cancer of the lower urinary tract. *Lancet* 1:1335-1337, 1971.

12. MacMahon B, Cole P, Brown JB, et al.: OEstrogen profiles of Asian and North American women. *Lancet* 2:900-902, 1971.

13. Hoover R, Cole P: Population trends in cigarette smoking and bladder cancer. *Am J Epidemiol* 94:409-418, 1971.

14. Cole P, MacMahon B: Attributable risk percent in case-control studies. *Br J Prev Soc Med* 25:242-244, 1971.

15. Allen DW, Cole P: Viruses and human cancer. *N Engl J Med* 286:70-82, 1972. Reprinted in *Ca—Cancer Journal for Clinicians* 23:127-136, 1973 and in *Diagnostic* 4:189-194, 1973.

16. Trichopoulos D, MacMahon B, Cole P: Menopause and breast cancer risk. *J Natl Cancer Inst* 48:605-613, 1972.

17. Cole P, Hoover R, Friedell GH: Occupation and cancer of the lower urinary tract. *Cancer* 29:1250-1260, 1972.

18. MacMahon B. Cole P: The ovarian etiology of human breast cancer. *Current Problems in the Epidemiology of Cancer, Lymphomas and Leukemias. Recent Results Cancer Res* 39:185-192, 1972.

19. Cole P: Epidemiology of Hodgkin's disease. *JAMA* 222:1636-1639, 1972.

20. MacMahon B, Cole P, Brown J: Etiology of human breast cancer: A review. *J Natl Cancer Inst* 50:21-42, 1973.

21. Hoover R, Cole P: Temporal aspects of occupational bladder carcinogenesis. *N Engl J Med* 288:1040-1043, 1973.

22. Cole P: Epidemiologic studies and surveillance of human cancers among personnel of virus laboratories. In Hellman A, Oxman MN, Pollack R (eds), *Biohazards in Biological Research.* Cold Spring Harbor Laboratory, New York, 1973.

23. Cole P: Hypotheses regarding the etiology of breast cancer. *Seventh National Cancer Conference Proceedings.* JB Lippincot Company, Philadelphi[a] 1973.

24. Cole P: A population-based study of bladder cancer. In Doll R, Vodpija I (eds), *Host Environment Interactions in the Etiology of Cancer in Man.* International Agency for Research on Cancer, Lyon, France, pp. 83-87, 1973.

25. Schmauz R, Cole P: Epidemiology of cancer of the renal pelvis and ureter. *J Natl Cancer Inst* 52:1431-1434, 1974.

26. Cole P: Epidemiology of human breast cancer. *J Invest Dermatol* 63:133-137, 1974.

27. MacMahon B, Cole P, Brown JB, et al.: Urine oestrogen profiles of Asian and North American women. *Int J Cancer* 14:161-167, 1974.

28. Cole P: Epidemiologic aspects of mammary Tumours. Proceedings of the Fifth International Symposium on the Biological Characterization of Human Tumours. *Excerpta Medica,* Amsterdam, 1974.

29. Dickinson L, MacMahon B, Cole P, Brown JB: Estrogen profiles of Oriental and Caucasian women in Hawaii. *N Engl J Med* 291:1211-1213, 1974.

30. Cole P: Morbidity in the United States. Chapter four in Erhardt CL, Berlin JE (eds), *A Review of Mortality and Morbidity in the United States.* American Public Health Association Monograph, Harvard University Press, 1974.

31. Cole P: Primary prevention of cancer. *Bull NY Acad Med* 51:75-79, 1975.

32. Cole P: Cancer of the lower urinary tract. In Schottenfeld D (ed), *Cancer Epidemiology and Prevention: Current Concepts.* Charles C. Thomas, Springfield, Illinois, 1975.

33. Gutensohn N, Li F, Johnson R, Cole P: Hodgkin's disease, tonsillectomy and family size. *N Engl J Med* 292:22-25, 1975.

34. Simon D, Yen S, Cole P: Coffee drinking and cancer of the lower urinary tract. *J Natl Cancer Inst* 54:587-591, 1975.

35. Cole P: Environmental factors in breast cancer: The epidemiologic evidence Proceedings of the XIth International Cancer Congress. *Excerpta Medica,* 1975.

36. Cole P, Goldman M: Occupation. Chapter 11 in Fraumeni JF (ed), *Persons at High Risk of Cancer: An Approach to Cancer Etiology and Control.* Academic Press, New York, 1975.

37. Ory H, Cole P, MacMahon B, Hoover R: Oral contraceptives and reduced risk of benign breast diseases. *N Engl J Med* 294:419-422, 1976.

38. Cole P: What should the physician ask? *Cancer* 37:434-436, 1976.

39. Morrison A, Cole P: Epidemiology of bladder cancer. In Prout GR (ed), *Urologic Clinics of North America,* Vol 3, No 1. WB Saunders, Philadelphia, February 1976.

40. Bunker JP, Donahue VC, Cole P, Notman M: Elective hysterectomy: Pro and con. *N Engl J. Med* 295:264-268, 1976.

41. Hoover R, Gray LA Sr, Cole P, MacMahon B: Menopausal estrogens and breast cancer. *N Engl J Med* 295:401-405, 1976.

42. Cole P, MacMahon B, Brown J: OEstrogen profiles of parous and nulliparous women. *Lancet* 2:596-599, 1976.

43. Grufferman S, Duong T, Cole P: Occupation and Hodgkin's disease. *J Natl Cancer Inst* 57:1193-1195, 1976.

44. Herbst A, Cole P, Colton T, et al.: Age-Incidence and risk of DES-related clear cell adenocarcinoma of the vagina and cervix. *Am J Obstet Gynecol* 128:43-50, 1977.

45. Grufferman S, Cole P, Smith P, Lukes RJ: Hodgkin's disease in siblings. *N Engl J Med* 296:248-250, 1977.

46. Cole P: Cancer and occupation: Status and needs of epidemiologic research. *Cancer* 39:1788-1791, 1977.

47. Cole P: Oral contraceptives and breast neoplasia. *Cancer* 39:1906-1908, 1977.

48. Gutensohn N, Cole P: Epidemiology of Hodgkin's disease in the young. *Int J Cancer* 19:595-604, 1977.

49. Sloan GM, Cole P, Wilson RE: Risk indicators of *de novo* malignancy in renal transplant recipients. *Transplant Proc* 9:1129-1132, 1977.

50. Isselbacher KJ, Cole P: Saccharin—the bitter sweet (editorial), *N Engl J Med* 296:1348-1350, 1977.

51. Cole P, Cramer D: Diet and cancer of endocrine target organs. *Cancer* 40: 434-437, 1977.

52. Cole P, Berlin J: Elective hysterectomy. *Am J Obstet Gynecol* 129:117-123, 1977.

53. Elwood JM, Cole P, Rothman K, Kaplan S: Epidemiology of endometrial cancer. *J Natl Cancer Inst* 59:1055-1060, 1977.

54. Peyster RG, Kalisher L, Cole P: Mammographic parenchymal patterns and prevalence of breast cancer. *Radiology* 125:387-391, 1977.

55. Herbst AL, Scully RE, Robboy SJ, et al.: Abnormal development of the human genital tract following prenatal exposure to diethylstilbestrol. In Hiatt H, Watson J, Winsten I (eds), *Origins of Human Cancer*. Cold Spring Harbor Laboratory, pp. 399-412, 1977.

56. Herbst AL, Cole P: Epidemiologic and clinical aspects of clear cell adenocarcinoma in young women. In Herbst AL (ed), *Intrauterine Exposure to Diethylstilbestrol in the Human. Proceedings of "Symposium on DES," 1977*. The American College of Obstetricians and Gynecologists, February 1978.

57. Cole P, Elwood JM, Kaplan S: Incidence rates and risk factors of benign breast neoplasms. *Am J Epidemiol* 108:112-120, 1978.

58. Hoover R, Bain C, Cole P, MacMahon B: Oral contraceptive use: Association with frequency of hospitalization and chronic disease risk indicators. *Am J Public Health* 68:335-341, 1978.

59. Cole P, Cramer D, Yen S, et al.: Estrogen profiles of premenopausal women with breast cancer. *Cancer Res* 38:745-748, 1978.

60. Cole P: Epidemiology. In Gusberg SB, Frick HC (eds), *Gynecologic Oncology*. The Williams and Wilkins Company, Baltimore, 1978.

61. Cole P, Morrison A: Basic issues in cancer screening. In Miller AB (ed), *Screening in Cancer*. UICC Technical Report Series, Vol 40, Union Internationale Contre le Cancer, Geneva, 1978.

62. Cooper J, Saracci R, Cole P: Describing the validity of carcinogen screening tests. *Brit J Cancer* 39:87-89, 1979.

63. Cole P: Screening—An epidemiologic approach in Fair R (ed.): *Selected Proceedings from the Symposium on Ocular and Systemic Disorders*, American Optometric Association, St. Louis, Missouri, 1978.

64. Cole P: The evolving case-control study. *J Chronic Dis* 32:15-27, 1979.

65. Grufferman S, Cole P, Levitan TR: Evidence against transmisison of Hodgkin's disease in high schools. *N Engl J Med* 300:1006-1011, 1979.

66. Johnson L, Driscoll S, Hertig A, et al: Vaginal adenosis in stillborns and neonates exposed to diethylstilbestrol and steroidal estrogens and progertins. *Obstet and Gynecol* 53:671-679, 1979.

67. Morrison A, Cole I . Epidemiology of urologic cancers. In Javadpour N (ed.), *Principles and Management of Urologic Cancer*. The Williams and Wilkins Company, Baltimore, 1979.

68. Austin H. Wynder E, Cole P: Breast cancer among Black American women. *Int J Cancer* 24:541-544, 1979.

69. Herbst A, Cole P, Norusis M, et al: An analysis of 384 registry cases of clear cell adenocarcinoma. *Am J Obstet Gynecol* 135:876-883, 1979.

70. Cole P: Some epidemiological aspects of cancer prevention (1979 Gordon Richards Memorial Lecture). *Cancer in Ontario*, 1979. Report of the Ontario Cancer Treatment and Research Foundation.

71. Herbst A, Scully R, Robboy S, et al: Diethylstilbestrol in pregnancy. *Proceedings of the Eppley Institute for Cancer Research* 1979:5-14. Omaha, Nebraska.

72. Cole P: Oral contraceptives and endometrial cancer (editorial) *N Engl J Med* 302:575-576, 1980.

73. Cole P, Morrison A: Basic issues in population screening for cancer. *J Natl Cancer Inst* 64:1263-1272, 1980.

74. Cole P, Merletti F: Chemical agents and occupational cancer. *J of Environ Path and Toxicol* 3:399-417, 1980.

75. Gutensohn N, Cole P: The epidemiology of Hodgkin's disease. *Sem Oncol* 7:92-102, 1980.

76. Trichopoulos D, Cole P, Brown JB, et al: Oestrogen profiles of primiparous and nulliparous women in Athens. *J Natl Cancer Inst* 65:43-46, 1980.

77. Cole P: Major aspects of the epidemiology of breast cancer. *Cancer* 46:865-867, 1980.

78. Cole P: Introduction. Chapter one in Breslow N, Day N (eds), *Statistical Methods in Cancer Research, Volume 1—The Analysis of Case-Control Studies*. IARC Scientific Publications No. 32, Lyon, France, 1980.

79. Hoar S, Morrison A, Cole P, et al: An occupation and exposure linkage system for the study of occupational carcinogenesis, *J Occ Med* 22:722-726, 1980.

80. MacMahon B, Andersen A, Brown J, et al: Urine estrogen profiles in European countries with high or low breast cancer rates. *European Journal of Cancer* 16:1627-1632, 1980.

81. Cole P: Analytic epidemiology. In *Proceedings of the 1980 International Symposium on Cancer*, pp. 25-34, Grune and Stratton, New York, N.Y., 1980.

82. Gutensohn N, Cole P: Epidemiology of Hodgkin's Disease. In Coltman CA, Golomb HM (eds) *Hodgkin's and Non-Hodgkin's Lymphomas*, Grune and Stratton, New York, 1980.

83. Gutensohn N, Cole P: Childhood social environment and Hodgkin's disease. *N Engl J Med* 304:135-140, 1981.

84. Cole P: Estrogens and progesterone in human breast cancer. *Banbury Report 8: Hormones and Breast Cancer*, pp. 109-113, Cold Spring Harbor, New York, 1981.

85. Cole P, Austin H: Breast self-examination: An adjuvant to early cancer detection. (editorial) *Am J Pub Health* 71:572-574, 1981.

86. Brisson J, Merletti F, Sadowsky N, et al: Mammographic features of the breast and breast cancer risk. *Am J Epidemiol* 115:428-437, 1982.

87. Brisson J, Sadowsky N, Twaddle J, et al: The relation of mammographic features of the breast to breast cancer risk factors. *Am J Epidemiol* 115:438-443, 1982.

88. Morrison A, Cole P: Urinary tract. Chapter 54 in Schottenfield D, Fraumeni JF (eds), *Cancer Epidemiology and Prevention*. W. B. Saunders Company, Philadelphia, PA, 1982.

89. MacMahon B. Trichopoulos D, Brown J, et al: Age at menarche, probability of ovulation and breast cancer risk. *Int J Cancer* 30:427-431, 1982.

90. MacMahon B, Trichopoulos D, Cole P, et al: Cigarette smoking and urinary estrogens. *N Engl J Med* 307:1062-1065, 1982.

91. Cole P: Epidemiologic clues to host factors in human carcinogenesis. In Armstrong B, Bartsch H (eds.), Host Factors in Human Carcinogenesis. IARC Scientific Publication No. 39, Lyon, France, 1982.

92. MacMahon B, Cole P, Brown J, et al: Urine estrogens, frequency of ovulation and breast cancer

risks: A case-control study in premenopausal women. *J Natl Cancer Inst* 70:247-250, 1983.

93. Greenberg R, Grufferman S, Cole P: An evaluation of space-time clustering in Hodgkin's disease. *J Chron Dis* 36:257-262, 1983.

94. Simard, A, Vauclair R, Cole P, et al: Pulmonary cytology in foundry workers. *J Chron Dis* 36:617-623, 1983.

95. Cole P, Austin H: The Role of the Epidemiologist. In Newell G (ed), *The Practice of Cancer Prevention in Clinical Medicine*, pp. 5-17, The Raven Press, New York, 1983.

96. Morrison A, Cole P, Maclure K: Epidemiology of urologic cancers. Chapter two in Javadpour N (ed), *Principles and Management of Urologic Cancer*, Second Edition. Williams and Wilkins, Baltimore, Maryland, 1983.

97. Cole P, Merletti F: Occupational Cancer. Chapter 15 in Bourke GJ (ed), *The Epidemiology of Cancer*. Croom Helm, Ltd., Kent, England, 1983.

98. Trichopoulos D, Yen S, Brown J, et al: The effect of Westernization on urine estrogens, frequency of ovulation and breast cancer risks: A study of ethnic Chinese women in the Orient and the U.S.A. *Cancer* 53:187-192, 1984.

99. Brisson J, Morrison A, Kopans D, et al: Height and weight, mammographic features of breast tissue, and breast cancer risk. *Am J Epidemiol* 119:371-381, 1984.

100. Scherr P, Gutensohn N, Cole P: School contact among persons with Hodgkin's disease. *Am J Epidemiol* 120:29-38, 1984.

101. Hochberg F, Toniolo P, Cole P: Head trauma and seizures as risk factors of glioblastoma. *Neurology* 34:1511-1514, 1984.

102. McCraw DS, Joyner RE, Cole P: Leukemia excess in a refinery population. *J Occ Med* 27:220-222, 1985.

103. Sohier R, Cole P, Montesano R: *Report on the IARC Research Training Fellowships Programme (1966-1984)*. IARC Internal Technical Report No. 86/002, WHO. Lyon, April 1986.

104. Austin H, Delzell E, Grufferman S, et al.: A case-control study of hepatocellular carcinoma and the hepatitis B virus, cigarette smoking, and alcohol consumption. *Cancer Research* 46:962-966, 1986.

105. Austin H, Cole P: Cigarette smoking and leukemia. *J Chron Dis* 39:417-421, 1986.

106. Austin H, Cole P, McCraw D: A case-control study of leukemia in an oil refinery. *J Occ Med* 28:1169-1173, 1986.

107. Mueller N, Swanson GM, Hsieh C, et al.: Tonsillectomy and Hodgkin's disease: Results from companion population-based studies. *J Natl Cancer Inst* 78:1-5, 1987.

108. Melnick S, Cole P, Anderson D, Herbst A: Rates and risks of diethyl-stilbestrol-related clear-cell adenocarcinoma of the vagina and cervix. An update. *N Engl J Med* 316:514-516, 1987.

109. Carpenter A, Flanders W, Frome E, et al.: Brain cancer and nonoccupational risk factors: A case-control study among workers at two nuclear facilities. *Am J Public Health* 77:1180-1182, 1987.

110. Austin H, Delzell E, Grufferman S, Levine R, Morrison AS, Stolley PD, Cole P: Case-control study of hepatocellular carcinoma, occupation, and chemical exposures. *J Occup Med* 29:665-9, 1987.

111. Austin H, Delzell E, Cole P: Benzene and leukemia: A review of the literature and a risk assessment. *Am J Epidemiol* 127:419-439, 1988.

112. Cole P: Saccharin and Bladder Cancer. Chapter nineteen in Gordis L (ed), *Epidemiology and Health Risk Assessment*. Oxford University Press, New York, 1988.

113. Butterworth CE, Hatch K, Cole P, et al: Zinc concentration in plasma and erythrocytes of subjects receiving folic acid supplementation. *Am J Clin Nutr* 47:484-6, 1988.

114. Waterbor J, Cole P, Delzell E, et al: The mortality experience of major-league baseball players. *N Engl J Med* 318:1278-80, 1988.

115. Cole P: Cancer: Risk factors and mortality trends. In Maulitz RC and Lear AC (eds), *Transactions & Studies*. Philadelphia College of Physicians. Philadelphia, 1988.

116. Delzell E, Austin H, Cole P: Epidemiologic studies of the petroleum industry. In Weaver NK (ed), *Occupational Medicine: State of the Art Reviews*, Volume 3. Hanley and Belfus, Inc., Philadelphia, 1988.

117. Austin H, Keil J, Cole P: A prospective study of cancer mortality in relation to serum DDT levels. *Am J Public Health* 79:43-46, 1989.

118. Delzell E, Macaluso M, Cole P: A follow-up study of workers at a dye and resin manufacturing plant. *J Occ Med* 31:273-278, 1989.

119. Wongsrichanalai C, Delzell E, Cole P: Mortality from leukemia and other diseases among workers at a petroleum refinery. *J Occ Med* 31:106-111, 1989.

120. Tamura T, Soong S, Sauberlich H, et al.: Evaluation of the deoxyuridine supression test using whole blood samples from folic acid-supplemented subjects. *Am J Clin Nutr* 51:80-86, 1990.

121. Hochberg F, Toniolo P, Cole P: Nonoccupational risk indicators of glioblastoma in adults. *Journal of Neuro-Oncology* 8:55-60, 1990.

122. Sharp G, Cole P: Vaginal bleeding and diethylstilbestrol exposure during pregnancy: Relationship to genital tract clear-cell adenocarcinoma and vaginal adenosis in daughters. *Am J Obstet Gynecol* 162:994-1001, 1990.

123. Sharp G, Cole P, Anderson D, et al: Clear cell adenocarcinoma of the lower genital tract. Correlation of mother's recall of diethylstilbestrol history with obstetrical records. *Cancer* 66:2215-2220, 1990.

124. Ibrahim MA, Bond GG, Burke TA, et al: The weight of the evidence on the human carcinogenicity of 2, 4-D. Howard School of Public Health, Program on Risk Analysis and Environmental Health. Final report, January 1990.

125. Cole P: The epidemiologist as an expert witness. *J Clin Epid* 44, Suppl. I, 35S-39S, 1991.

126. Macaluso M, Delzell E, Cole P, Wongsrichanalai C, Cowles S: Validity of a mortality study based on a corporate health surveillance system. *J Occup Med* 33:1180-1186, 1991.

127. Cole, P: The value of an indirect index to select groups for a health education message (editorial). *Epidemiology* 2:401-402, 1991.

128. Butterworth C, Hatch D, Macaluso M, Cole P, et al: Folate deficiency and cervical dysplasia. *JAMA*. In press.

129. Chen F, Cole P, Mi Z, Xing L: Dietary zinc and esophageal cancer mortality in Shanxi, China. Submitted.

130. Sathiakumar N, Delzell E, Cole P, Austin H: A follow-up study of agricultural chemical production workers. Submitted.

131. Sharp G, Cole P: Identification of risk factors for diethylstilbestrol-associated clear cell adenocarcinoma of the vagina: similarities to endometrial cancer. Submitted.

132. Funkhouser E, Cole P: Declining mortality for cancer of the rectum, 1940-1985. Submitted.

133. Funkhouser E, Cole P: Mammographic patterns and breast cancer risk: Summary relative risk estimates. Submitted.

134. Funkhouser E, Waterbor J, Cole P, Rubin E: Mammographic patterns, breast cancer risk factors and a breast cancer risk score. Submitted.

135. Butterworth CE, Hatch KD, Soong S, et al: Oral folic acid supplementation for cervical dysplasia: a clinical intervention trial. Submitted.

136. Liu T, Wilson NP, Craig CB, et al: Evaluation of three nutritional assessment methods in a group of women. Submitted.

137. Cole P and Hovinga M: Hormone residues in meat products. *Prevention*. In press.

138. Jones TL, Shih CH, Thurston DM, Ware BJ and Cole P: Residential mobility as an explanation for associations of wire configuration codes with childhood cancer. Submitted.

139. Cole P, Delzell E and Acquavella J: Exposure to butadiene and lymphatic and hematopoietic cancer. Submitted.

Letters and Book Reviews

1. Cole P: Cohorts and conclusions. *N Engl J Med* 278:1126-1127, 1968.

2. Cole P, MacMahon B: Urinary oestrogen profiles and aetiology of breast cancer. *Lancet* 2:153, 1970.

3. MacMahon B, Cole P, Newell GR: Hodgkin's disease: one entity or two? *Lancet* 1:240-241, 1971.

4. Cole P, Hoover R: Letters to the editor. *J Natl Cancer Inst* 46:1111-1113, 1971.

5. Cole P, et al: Tonsillectomy and Hodgkin's disease. *N Engl J Med* 288:634, 1973.

6. MacMahon B, Cole P, Brown JB: Estrogen profiles and breast cancer. *N Engl J Med* 292:974-975, 1975.

7. Gutensohn N, Cole P, Li F: Hodgkin's disease and sibship size. *N Engl J Med* 292:1025-1038, 1975.

8. Cole P: Costs and benefits of hysterectomy. *N Engl J Med* 295:1086, 1976.

9. Grufferman S, Cole P, Smith P, Lukes RJ: Reasons for familial aggregation in Hodgkin's disease. *N Engl J Med* 296:940-941, 1977.

10. Gutensohn N, Cole P: Response to Vianna et al. *Int J Cancer* 20:633-634, 1977.

11. Gutensohn N, Cole P: Immunity in Hodgkin's disease. *Ann Intern Med* 91:316-317, 1979.

12. Gutensohn N, Cole P: Letter to the editor. *N Engl J Med* 304:1171, 1981.

13. Merletti F and Cole P: Detection bias and endometrial cancer. *Lancet* ii:579-580, 1981.

14. Cole P, Austin H: Reply to letter on breast self-examination. *Am J Pub Health* 71:1277, 1981.

15. Cole P, Austin H: Reply to letter on breast self-examination. *Am J Pub Health* 72:499-500, 1982.

16. MacMahon B, Trichopoulos D, Cole P and Brown J: Smoking and urinary estrogens. Letter to the Editor. *N Engl J Med* 308:591, 1983.

17. Austin H, Cole P: *Early Detection of Testicular Cancer.* (Book Review) *Chemother* 28:311, 1982.

18. Merletti F, Cole P: UICC Technical Report Series, Vol. 41, *Cancer Risks by Site.* (Book Review). *Chemother* 28(4):312, 1982.

19. Grufferman S, Cole P: Interaction between sex and HLA type and Hodgkin's Disease. *Arch Neurol* 41:816, 1984.

20. Austin H, Delzell E, Cole P, Grufferman S, Levine R, Morrison AS and Stolley PD: Reply to a letter regarding a case-control study of hepatocellular carcinoma. *Cancer Res* 48:654-655, 1987.

21. Austin H, Delzell E, Cole P: Benzene and Leukemia. Letter to the editor. *N Engl J Med* 317:1027-28, 1987.

22. Cole P: Carpenter D and Ahlbom A: Comments on electromagnetic fields and cancer in human beings, especially children. Letter to the editor (by invitation). *Forum for Applied Research and Public Policy*, 1989.

23. Austin H, Cole P: Reply to letter regarding cigarette smoking and leukemia. *J Clin Epidemiol* 42:1027-28, 1989.

[Plaintiff's Exhibit 4]

SMOKING AND SMALL CELL LUNG CANCER

Histology

Small cell lung cancer (SCLC) constitutes about 15%-20% of all lung and bronchial cancers (1). It develops predominantly in the central and intermediates sections of the bronchial system. (Squamous cell carcinoma tends to occur somewhat more peripherally.) SCLC appears on light microscopy as small, spindle-shaped or lymphocyte-like tumor cells, poor in cytoplasm and with bare nuclei. Three subtypes have been described (2):

1. Giant cell type—uniform tumor, round nucleoli and scarcely developed stroma.
2. Intermediate cell type—large, spindle—or oval shaped nucleoli; also polygonal nuclei. Cytoplasm is more developed and pseudoductal structures can be found.
3. Combined type—has small numbers of squamous cells and/or adenocarcinoma structures in addition to the predominating small cell structures.

Both SCLC and squamous cell carcinoma are Type I tumors under the Kreysberg classification system.

Association with smoking

SCLC is strongly associated with smoking. Estimates of the relative risk (odds ratio) varies from 5.0-42.5 for current smokers (3-7). These estimates are comparable with the magnitude of association of squamous cell lung cancer with smoking. The attributable risk for smoking (among smokers) is estimated at 89%-97% (3,4). Similar levels of risk have been shown for both male and female smokers. Weaker associations (RR = 1.6-3.0) have been shown for passive smokers (7-9). A convincing dose-response exists between the amount of cigarettes

smoked or years of smoking and the risk of SCLC. The risk of developing SCLC decreases with smoking cessation but the rate of decrease is less pronounced compared to squamous cell lung cancer. The relative and the absolute risk of developing SCLC plateaus 20-39 years after start of smoking (3). However, the risk of SCLC is increased among smokers as early as 5-9 years after start of smoking.

Prognosis

The prognosis for SCLC is probably slightly worse than that or squamous cell carcinoma.

REFERENCES

1. Muller KM, Mene R. Small cell cancer of the lung: Pathological anatomy. IN: Seeber S, ed. Small cell lung cancer. Springer-Verlag, New York 1985.
2. World Health Organization (WHO). Histological classification of lung tumors. WHO, Geneva 1981.
3. Jedrychowski W, Becher H, Wahrendorf J, Basa-Cierpialek Z, Gomola K. Effect of tobacco smoking on various histological types of lung cancer. J Cancer Res Clin Oncol 1992; 118:276-282.
4. Morabia A, Wynder EL. Cigarette smoking and lung cancer types. Cancer 1991; 68:2074-2078.
5. Brownson RC, Chang JC, Davis JR. Gender and histologic type variations in smoking-related risk of lung cancer. Epidemiology 1992; 3:61-64.
6. Osann KE, Anton-Culver H, Kurosaki T, Taylor T. Sex differences in lung-cancer risk associated with cigarette smoking. Int. J Cancer 1993; 54:44-48.
7. Svensson C, Pershagen G, Klominek J. Smoking and passive smoking in relation to lung cancer in women. Acta Oncologica 1989; 28:623-629.

8. Correa P, Pickle LW, Fontham E, Lin Y, Tockman MS. Passive smoking and lung cancer. Lancet 1983; 2:677.
9. Sandler DP, Everson RB, Wilcox AS. Passive smoking and adulthood and cancer risk. Am J Epidemiol 1985; 121:37.

PHILIP COLE, MD, DrPH
August 2, 1993

UNITED STATES DISTRICT COURT
NORTHERN DISTRICT OF GEORGIA
ATLANTA DIVISION

(Title Omitted in Printing)

The deposition of WILLIAM J. WADDELL, M.D., taken pursuant to Notice and agreement for cross-examination, discovery and any other purpose allowed by law and pursuant to the Federal Rules of Civil Procedure and the Federal Rules of Evidence; all formalities waived, including the reading and signing of the deposition; before Joyce S. Oglesby, Certified Court Reporter and Notary Public in and for the State of Georgia; commencing at 10:10 a.m., Tuesday, October 19, 1993 at 4000 One Peachtree Center, 303 Peachtree Street, Atlanta, Georgia.

* * * *

[4] WILLIAM J. WADDELL, M.D.

was called as a witness herein and, having been first duly sworn, was examined and deposed as follows:

* * * *

[17] Q. No. 8 is a two-volume collection entitled Medical Records of Robert Kevin Joiner.

A. That's correct.

Q. Have you reviewed these?

A. I went through those, yes.

Q. When you say went through, tell me the level of—

A. I did—I did not read them carefully. I just skimmed through them to see if there was anything in there that might be pertinent.

Q. Was there anything pertinent?

A. Well, the diagnosis is pertinent, his smoking history and drinking, things like that.

Q. What do you understand the diagnosis to be?

A. Small cell carcinoma of the lung.

Q. And you mentioned there was a smoking history?

A. That's correct.

Q. What do you understand that to be?

A. Well, it's highly variable in there. It varies anywhere from 1 pack a day for 8 years to 2 packs a day for 15 years, so there must be something. It's hard to say exactly what it is.

* * * *

[24] Q. When did you make your opinion in this case? How long had you had the case before you reached your conclusions on it?

A. Well, once I learned that it was cancer of the lung, I had trouble believing it was caused by PCBs, and so I would guess from the very—from very early on [25] because I've sort of kept up with PCBs. My interest—My research interest is in chemical carcinogenesis, and I'm interested in what kind of chemicals do cause cancer, and I know that the literature does not show that there's any causal relationship between PCBs and lung cancer.

* * * *

[32] Q. And that methodology of using animals is, in fact, scientifically accepted in the community as a whole?

A. The method is accepted—is accepted and is appropriate and proper, but the information cannot be blindly extrapolated to humans. It must be extrapolated with great caution.

* * * *

[38] Q. Adipose tissue.

A. In my adipose tissue, I would guess that one part per million, something like that.

Q. What do you think the national averages are?

A. Well, the NHATS report is the best one. That's the most comprehensive of levels throughout the country, in which they took autopsy samples and analyzed them for a wide variety of chemicals, and I think the extrapola-

tion was in—in the late '80s that 95 percent of the people would have at least one part per million, something like that.

* * * *

[41] Q. Do you know of a number that you can say that is the safe dose [of PCBs]?

A. Well, are you trying to get at the calculations that have been made by the EPA and others as far as what they consider a safe dose in—

Q. No, sir, I'm trying to get your opinion as to what you consider a safe dose.

A. My opinion?

Q. Yes, sir.

A. Well, my opinion is that—that the doses to which people have been exposed currently are all safe, including those in—in manufacturing and industry because we don't see any health effects from those. There are no health effects that are consistent and [42] reproducible that have been demonstrated for any exposures currently. The only adverse affects were those early on in the '30s or so in which they had apparently massive exposures and yellow atrophy of the liver.

Q. How about furans, polychlorinated dibenzofurans, what would be a safe level of contamination?

A. Sir, I'd have—I'd have to answer the same way. We don't have specific information on that.

Q. You don't consider the Yusho studies to show some correlation between the adverse human health effects and furans?

A. We don't know what the dose was. There's no way to calculate the dose because the technology at the time was not good enough to say how many furans and how many chlorophenols and other things were in there, so there's no way to calculate a dose.

* * * *

[46] Q. But your opinion with respect to dioxins and furans and PCBs is all the same, that is that the levels to which people have been exposed have not caused any human health effects?

A. That's correct, the ordinary levels that people have been exposed, yes, they're all the same.

Q. And you believe there are safe levels of furans and dioxins, but you're not prepared to tell me today what they are?

A. Absolutely, there are safe levels.

Q. And what are they?

A. Well, the levels to which people are exposed today and have in their blood and fat today are safe because there's no evidence of adverse health effects from these.

* * * *

[54] Q. You tell me that you—What do you do to maintain currency in the PCB world?

A. I keep up with the literature.

* * * *

[57] Q. Okay. Why is it that you keep up with PCBs?

A. Well, it's an interesting group of chemicals [58] because they're not nongenotoxic, and there's a widespread interest in them. I keep up with those and other nongenotoxic chemicals such as phenobarbital, and the—the analogies between those. You know, there are certain chemicals that offer us some clues about the mechanism of carcinogenesis.

And phenobarbital, of course, is another interest of mine from medical school days for the reasons I've just. . . You know, this—this clinical treatment of phenobarbital poisoning that I developed is still in clinical use today, so, you know, I've kept up with it, kept an interest in phenobarbital. And since it is not carcinogenic and is in widespread use and yet it is carcinogenic to animals, it makes a very good surrogate for looking at the differences between cancer in animals and in humans. And the same thing is true of the chlorinated hydrocarbons which cause cancer in animals but not in humans.

And so, the—the question is why do the compounds cause cancer in humans—in animals and not in humans. Something is wrong with the animal model, and so what

is wrong with the animal model? That's why I keep up with them.

* * * *

[59] Q. Well, I want the ones that in any way support your opinions in this case that Mr. Joiner does not have cancer, nor does any other human on earth have cancer because of polychlorinated biphenyls.

A. Fair enough. And I think that the answer is that the—the— There are several answers to that. The— The studies in experimental animals that show cancer from PCBs in animals are enormous doses. They're at the maximum-tolerated dose, as are the substance—many of the other substances that cause cancer in animals and not in humans. They're given at the maximum-tolerated dose.

The maximum-tolerated dose was devised as an expedient 30 years ago to try to screen for compounds that might be carcinogenic, and those were in our early days of understanding of the mechanism of carcinogenesis when we thought that there were two kinds of compounds, those that were safe and those that were—caused cancer. It was a naive understanding of cancer, which [60] today has proved wrong. In other words, we can't separate them that way. It looks more and more like it's merely the dose that causes the cancer. And so, the question is: Why does a high dose? Because more than half of all the compounds, the thousands of compounds that have been tested in the NCI Assay at the maximum-tolerated dose cause cancer in rodents, and the cancer is a liver cancer. And yet, if we look at liver cancer in humans, it's actually declining at a time when humans have been continually exposed to these same compounds.

If you plot the incidents and death rate from cancer of the liver, it's declining in the United States and steadily. It's one of the lowest ones. And the evidence is that virtually all liver cancer is due to cirrhosis. And the debate among hepatologists now is that there is not

a case, documented case of liver cancer in humans that does not have cirrhosis, either from a hepatitis virus or some other reason of cirrhosis. It's the damage from cirrhosis from either alcohol or viral infection that causes liver cancer in humans. And yet, virtually all of the cancers in experimental animals are liver cancers.

So, we have to come to the obvious conclusion that the experiments done with NCI Assay in rodents that produce liver cancer have no [61] predictability for human cancer. And the question as far as a mechanistic explanation that those of us who are interested in mechanism are toying with is what does explain that the high dose causes liver cancer in rodents.

Q. If it's your opinion that the study of cancer in animals cannot be extrapolated at all to humans, is it just animal cruelty to continue to cause animals in laboratories to get cancer?

A. At the maximum-tolerated dose, it's my opinion that it's worthless, that we now have enough database to where we need to stop doing that, that the database is adequate now of the maximum-tolerated dose in rodents, and I'm not alone in this feeling, that the further ex— further experiments on rodents at the maximum-tolerated dose will add nothing to our information.

Q. Is your feeling widely accepted in the scientific community and do you find—

A. It's accepted by very—some very prominent scientists.

Q. Is it widely accepted in the scientific community?

A. I—It depends on who's voting.

Q. In other words, this opinion, like some of [62] the other ones you've told me about is—there is indeed a split decision?

A. Well, let's put it like this, Michael: There—There are a number of senior scientists that see it the same way I do. They probably numberwise are in the minority. There are a lot of young scientists that are going with

the flow and have—do not have the perspective. So if you actually took a vote, the senior scientists that have it in perspective that think it's of no value, would be in the minority. Yet, to my way of thinking, those are the scientists whose votes count more.

* * * *

[64] Q. Lucy Anderson's done two studies.

A. She's done many more than that.

Q. That I'm interested in that we've talked about with—

A. Okay.

Q. —Dr. Robertson and Dr. Schecter. Are you familiar with those two?

A. Those two, right.

Q. About mice and lungs.

A. Fetal—Newborn mice, suckling mice.

Q. Do those studies have any validity at all in human beings?

A. No.

Q. Why not?

A. Well, because it's only in newborn mice, and the enzyme systems, the P450 systems in newborn mice are not developed yet. They don't develop until about the time of weaning. They wean at about three weeks of age, and these studies were all done before three weeks of [65] age, in which the enzyme systems are not developed yet. That's—That's one thing, so that inducing agents during that period of time probably have no relevance to inductions of those enzymes as an adult. And there is the obvious difference that these are just strains of mice, and we don't know what those sorts of—how we can extrapolate those because they're unique kinds of experiments.

Q. Speaking of infants, if you have a grandchild, would you allow them to play in a room that had air-

borne concentrations of 50 parts per million, what the EPA would consider a PCB-contaminated room?

* * * *

A. I have no problem with that as far as that causing cancer. Let's put it like this: There is no evidence that PCBs cause cancer, no matter what the age of exposure is. Otherwise, we would be seeing an increase in cancer of the lung, other than that caused by smoking, and we don't see that. The cause of lung cancer is smoking, and that's the only identified thing that we [66] have.

* * * *

[67] Q. In fact, you think that the ban on PCBs is not necessary; is that correct?

A. The ban on PCBs is probably what—was probably premature. If we can find good substitutes, there's no—there's no problem with banning anything. I think that the main problem with the PCB question is the fanatical attention we're paying to cleaning up PCB [68] contamination, which—which is totally unnecessary.

* * * *

[79] Q. And that's consistent that you do not worry about PCBs in humans at all?

[80] A. That's correct.

Q. And that would be both for people like us who wear neckties and people who work in factories where they make capacitors?

A. The levels that people are exposed to now, there's no evidence at all that they have any adverse health effects.

Q. What about those studies from Bloomington, Indiana that Greg Steele and some other people did on high levels of brain cancer among capacitor workers at that plant?

A. What about them?

Q. Is that some evidence that it may have an effect at least on that particular kind of cancer?

A. No, because the epidemiology studies have no consistency and no specificity. They vary from one study to another, and they're—none of them are significant. We have the Hill criteria which we have to use for epidemiological studies.

Q. What's the Hill criteria?

A. Strength of the association, specificity of association, consistency of the association, temporality of the association, dose response, experiment, analogy. Those are criteria that we would have to use in epidemiological studies, and the main ones are [81] consistency and strength. All of these studies are very weak associations, and there is no consistency. If it causes brain cancer in one study, malignant melanoma in another, lymphomas in another, you've got to ask the question if the strength is very low and the power is such that we can't say that it really does it, on balance, it's noise in the data, and that's what we have.

Q. Could it be that PCBs and their—And when I've been talking about PCBs, I've been including contaminants such as furans at levels that would be—

A. Of course.

Q. Have you?

A. Yes, of course.

Q. Would it be a possibility that the variation in the findings bring cancer in one cohort and a malignant melanoma in another cohort, liver disease in another cohort that concern you so that they're not all consistent, isn't there some environmental factor for each cohort that is initiating the cancer and that the PCBs just come along and promote it in some efficient fashion? Why isn't that a possibility?

A. Well, because there's no strength to those associations. All of them, even—All of them are either nonsignificant by statistical calculation or those that are significant are barely significant, meaning that [82] the 95 percent confidence of the lower level—the lower limit of

the 95 percent confidence to normal is barely over 1. We have seen enough studies in which we have epidemiological studies that still is within the noise of the data.

* * * *

[83] Q. What do you think about the individual susceptibility, a given person may have a higher potential because of their own personal genetic makeup to be affected by a chemical than other members in the group. Is that possible?

A. Anything is possible.

Q. I mean, are we all, as you said, regularly exposed to things that are cancerous at least in rats? Are we all also exposed to things that we know are cancerous in humans, like second-hand cigarette smoke, car exhaust?

A. I think the second-hand cigarette smoke is debatable, whether that causes cancer.

Q. Well, first-hand cigarette smoke?

A. First-hand cigarette smoke, there is no doubt. I don't think that there is any reasonable scientist that would deny that cigarette smoking causes lung cancer.

* * * *

[89] Q. Well, assume that this transformer may be fully repaired or it may eventually get hit by lightning and spill all over somebody's yard. Should they be warned that there's any health hazard whatsoever associated with it? Should they be told not to inhale, ingest or absorb any of that chemical? Or is the risk simply just so low that you don't think they need any warning whatsoever?

A. Well, let's just say that the risk is from the—the track record that we have on PCBs in fluids and transformers is such that no one has been injured—has had health effects from those exposures.

Q. So, therefore, based on the history that you know today and what you believe the history to be in the future or what the future holds, there would be no need to warn against inhalation, ingestion or absorption of that chemical; is that correct?

A. Based on the epidemiology we have now and the track record we have and what exposures have been, there is no evidence that there would be a situation [90] which there would be adverse health effects. Anything is toxic if the dose is high enough. You can kill yourself by drinking too much water.

* * * *

[93] Q. With respect to cancer, what's an initiator and what's a promoter?

A. Well, the current theory, hypothesis is that an initiator is something that attacks the DNA, that alkylates or in some way changes the DNA in a cell. And a promoter is something that promotes the cell's dividing once it has been initiated to make it grow more.

Q. Is cigarette smoke an initiator?

A. It is.

Q. Is it a promoter?

A. Probably.

* * * *

[97] Q. Do you think that the concept of initiation and promotion has anything at all to do with Mr. Joiner's onset of lung cancer?

A. I would have to say we don't know. We—We don't know, but I think that we can answer as far as the specific case of Mr. Joiner that promotion—that we have no information that would lead us to believe that the PCBs promoted the initiation from the—from the cigarette smoke and had any significant effect on his development of lung cancer.

Q. Do you think that PCBs can promote any cancer?

A. No. There no evi—information that they can promote any cancer in humans.

[98] Q. Do you think there's information that they have promoted cancer in animals?

A. Under specific circumstances, like the Lucy Anderson papers. There are some circumstances in which they

have been interpreted to be promoters in experimental animals.

Q. Do you think that they can initiate cancer in animals?

A. There's no evidence that they can initiate cancer. They're called nongenotoxic.

Q. With respect—Can furans initiate cancer?

A. No.

Q. Dioxins?

A. No.

Q. All of those three are promoters?

A. All those in experimental animals can be promoters. They can also be inhibitors.

Q. Are furans promoters in humans?

A. In humans?

Q. Yes.

A. There's no evidence that they are.

Q. Are dioxins promoters in humans?

A. No evidence that they are.

* * * *

[99] Q. [. . .] [100] Describe for me the science of toxicology.

A. The science of toxicology is the study of the adverse effects of chemicals.

Q. Describe the scientific evidence that a toxicologist considers when—And I want these answers to be relevant to the PCB case and your analysis and opinions in this case involving Mr. Joiner, please.

What kind of evidence would you consider as a toxicologist?

A. What kind of evidence I would consider? I would consider primarily epidemiological evidence.

Q. Epidemiological evidence concerning populations of human beings?

A. Populations of human beings. That's the definition of epidemiology.

Q. What role does human epidemiological studies play —do human epidemiological studies play in the determination of the toxicity of any substances?

A. Epidemiology plays a—a prominent role because that's the final species to—in which we're interested. Experimental animals cannot predict; they only give us some clues and the suggestion of ways to go in humans, but ultimately, the epidemiological studies [101] are those on which we must rely.

Q. What do you understand to be the state-of-the-art causes of human cancer or have we already discussed that moments ago?

A. State-of-the-art causes of human cancer? That's pretty broad. What do you want me to talk about?

Q. Well, Mr. Freeman wrote me the question. I'm just trying to see if I understand it.

It says here that you "would be able to testify as to the state of the art as to what is known regarding the causes of cancer in humans, generally." What is your opinion on that?

A. Well, there are a few known causes of cancer in humans. One is cigarette smoking. Another one is hepatitis virus. There are a few known causes of cancer in human.

Q. Do you believe that cancer in humans is growing at a—as the percentage of the population is at the rate it's growing?

A. The incidents [*sic*—incidence] and death rate from cancer is increasing currently only because of the incease in lung cancer. If one takes the lung cancer out of the cancer statistics, then there is an overall decine in cancer in the United States.

* * * *

[109] Q. What do you understand that Mr. Joiner suffers from?

A. Small cell carcinoma of the lung.

Q. What do you believe caused it?

A. Smoking tobacco.

Q. And what was his statistical chance of being diagnosed with small cell carcinoma at the age of onset?

A. I don't have a number, but it's low because he developed it at a relatively low age, and the number of people that develop cancer of the lung from smoking decreases with decreasing age.

Q. Well, you saw in Dr. Robertson's testimony that he had introduced or had with him a study that had some statistics. Do you think that they would be off base?

A. The stat—Would the statistics be off base or the interpretation be off base?

Q. The statistics.

[110] A. I don't know specifically what the statistics were, but they probably were the sera data from the National Cancer Institute. That's what everybody's using, and they probably show a very low incidence at—at 20 years of age, 30 years of age with increasing at each 5-year interval. So what? We know that there are people that have—that develop cancer in their 20's from smoking for a few years. Some people are simply more susceptible than others. We do not know the reason. The only thing we can infer is that there is a genetic predisposition in some people.

* * * *

[111] Q. Well, the way that Lucy Anderson did it, it appeared to be a promoter there; is that correct?

A. Well, but that was in—that was in suckling mice whose enzymes are totally different, totally different. We published papers on this many years ago about how the enzymes do not mature in mice until the—essentially the time of weaning, which is about three years—three months —three weeks of age. And so, if you take suckling mice, you can get all sorts of curious effects.

Q. At least in that particular example, it did appear to be a promoter; is that correct?

A. In that particular situation, but Mr. Joiner is not a suckling mouse.

* * * *

[112] Q. Have you ever heard of the World Health Organization?

A. Yes, sir.

Q. Are you familiar with their recent thing that Dr. Teitelbaum discussed, their publication called the International Program on Chemical Safety?

A. I'm familiar with the IPCS.

Q. Okay. Are you familiar with the one on Polychlorinated B[i]phenyls and Terphenyls, Second Edition?

A. I can't recall.

Q. Well, it's just come out in the last month or two, if you've seen it. It's a red book.

A. I don't recall seeing it, no.

Q. On page 478 of that publication, it says, "Taking the combined evidence from human and experimental animal studies, the IARC group concluded that PCBs are probably carcinogenic for humans." Then it has a parenthesis, IARC, 1987, closed parenthesis, period.

What is your reaction to that phrase?

[113] A. That phrase is just another way of saying that it's been found to be carcinogenic in animals. That's all that—I've been to the IARC discussion, I've been there a couple times and participated in those workshops, and that's all that means. If it's found to be carcinogenic in animals, then it's presumed to be carcinogenic in humans, but there is no evidence that it's carcinogenic in humans, and that document will so state.

Q. So, the word "probably" carcinogenic for humans doesn't mean that at all?

A. That does not in the sense that you're talking about.

* * * *

[116] Q. Did you read Dr. Schecter's deposition?

A. I read the excerpts prepared by Teitelbaum's staff.

Q. What did you think of his thoughts?

A. Well, I—I completely disagreed with his interpretation of what happens when you lose body fat. I think he's just going in exactly the wrong direction, and I think all the scientific information will back me up on that.

Q. What about his other opinions, that PCBs are not good for humans and nor are furans or dioxins?

A. I think he's exaggerating the information we have available.

Q. Mr. Freeman handed me this morning the [117] following with respect to the opinions that he expects you to provide at the trial: Number one, "That there is no scientific evidence that PCBs are an initiator, co-carcinogen or promoter of, one, small cell carcinoma of the lung in humans or, two, any malignancy in the human lungs," Is that your opinion?

A. That's correct.

Q. And we've talked about how you arrived at that opinion already, haven't we?

A. I think we have.

Q. Number two, "There is no scientific basis upon which to conclude that Mr. Joiner's small cell carcinoma of the lung was caused by his exposure to PCBs"?

A. That's correct.

Q. And it would be, regardless of the dose, you just don't think that there's any way you can get lung cancer from PCBs; is that correct?

A. That's correct.

Q. So, we're not arguing about the level of exposure because there is no level that you believe could cause lung cancer; is that correct?

A. There is no information in humans that PCBs cause cancer of the lung at whatever dose. There is—

Q. What about furans?

[118] A. No information.

Q. Dioxins?

A. No—No evidence that they do.

Q. Number three, "The only recognized cause of small cell cancer of the lung is exposure to cigarette smoke."

A. That's correct.

Q. And you've talked to me about that already; is that correct?

A. Right, I think so.

Q. Is it your further opinion that a small cell cancer caused by cigarette smoke could not be promoted by any other chemical?

A. In humans, I think there is no information that it would be promoted by anything.

* * * *

[119] Q. Number four, "Mr. Joiner has not received a toxicologically-significant dose of PCBs. His adipose tissue level is well within established ranges for concentrations reported in the general population."

That's part of your opinion?

A. It is.

Q. And we've talked about that a little bit?

A. I think we have.

Q. What is a toxicologically-significant dose of PCBs?

A. Well, it's a dose—I can't tell you what it would be, but it certainly would be above that.

* * * *

[123] Q. The second portion of this number four was, "His adipose tissue level is well within established ranges for concentrations reported in the general population."

You told me earlier that you would expect that to be around one part per million?

A. Yes, and because I think the NHATS thing said that 95 percent of the population, and I think that was in 1988 or '89. I can't remember. The NHATS thing was discontinued about '84 or '85, but they had enough information to make projections about what the levels were and how they were increasing in numbers of people. I—I've got a slide on that, but I—

Q. Are you familiar with the work done on these PCB levels that was published in "Chemisphere" by some scientists at the CDC; they did about 5,000 samples?

A. I probably have seen that.

* * * *

[124] Q. This last thing that we talked about, number five, "There's no scientific basis to believe that weight loss diminishes concentration of PCBs in the adipose tissue." We've talked about that, have we not?

A. Yes.

* * * *

[Plaintiffs Exhibit 1]

WILLIAM JOSEPH WADDELL

Date and Place of Birth:

March 16, 1929, Commerce, Georgia, USA

Marital Status: Married, 3 Children

Social Security Number: 723-05-5162

Office Address and Phone Number:

Department of Pharmacology and Toxicology
University of Louisville School of Medicine
Louisville, Kentucky 40292
502/588-5141

Home Address and Phone Number:

6604 Gunpowder Lane
Prospect, Kentucky 40059
502/228-4220

Education:

A.B., (Chemistry), University of North Carolina, 1951
M.D., University of North Carolina, 1955

Fellowships:

Public Health Service Postdoctoral Research Fellow, Department of Pharmacology, University of North Carolina, 1954-1958
National Institutes of Health Special Fellow, Department of Pharmacology, Royal Veterinary College, Stockholm, Sweden, 1965-1966
Scholar, Sloan Foundation, Health Sciences Consortium, 1974-1975

Academic Appointments:

Assistant Professor of Pharmacology, School of Medicine, University of North Carolina, 1958-1962
Associate Professor of Pharmacology, School of Medicine, University of North Carolina, 1962-1972
Associate Professor of Oral Biology, Dental Research Center, University of North Carolina, 1967-1969
Professor of Oral Biology, Dental Research Center, University of North Carolina, 1969-1972
Professor of Pharmacology, College of Medicine, University of Kentucky, 1972-1976
Professor of Oral Biology, College of Dentistry, University of Kentucky, 1972-1976
Adjunct Professor of Pharmacology, School of Medicine, University of Louisville, 1972-1976
Adjunct Professor of Pharmacology, College of Medicine, University of Kentucky, 1977-
Professor of Pharmacology and Toxicology, School of Medicine, University of Louisville, 1977-
Visiting Professor of Pharmacology and Toxicology, Schools of Medicine and Pharmaceutical Sciences, Showa University, Tokyo, Japan, November 1983-May 1984

Honors:

Centennial Alumnus Distinguished Visiting Professor, School of Medicine, University of North Carolina, February, 1979

Administrative Appointments:

Associate Division Director, Center for Research in Pharmacology and Toxicology, University of North Carolina, 1966-1967
Associate Director, Dental Research Center, University of North Carolina, 1968-1972
Director of Graduate Studies in Pharmacology, University of Kentucky, 1974-1976

Chairman, Department of Pharmacology and Toxicology, University of Louisville, 1977-

Society Memberships:

American Physiological Society, 1973-
American Society for Pharmacology and Experimental Therapeutics, 1958-
American Teratology Society, 1968-
Association for Medical School Pharmacology, 1977- (Secretary 1986-1988; President 1988-1990)
Sigma Xi, 1958-
Society for Experimental Biology and Medicine, 1956-
Society of Toxicology, 1978-
Ohio Valley Chapter of Society of Toxicology, 1983- (President 1983-84)
Royal Microscopical Society, 1986-
International Society for the Study of Xenobiotics, 1989-
Fellow, Academy of Toxicological Sciences, 1992-

Consultant:

Research Triangle Institute, 1965-1972
Block Drug Company, 1968-1972
Becton, Dickinson and Company, 1971-1972
Baxter-Travenol Laboratories, 1974-1980
Faculty of Medicine, University of Kuwait, 1977-
Baby Products Company, Johnson and Johnson, 1977-1982
Procter and Gamble, 1978-
Allied Chemical, 1978-1982
American Cyanamid, 1981-1987
Dow Chemical, 1981-1986
R.J. Reynolds Industries, Inc., Scientific Advisory Board, 1985-1988 (Chairman 1987-1988)
CA Blockers, Inc., 1987-1989
Grain Processing Corporation, Inc., 1988-
Norwich Eaton Pharmaceuticals, Inc., 1988-
Distilled Spirits Council of the United States, Inc., 1989-

Board of Directors:

Computer Assisted Teaching Systems (CATS) Consortium, 1977-1983
Pharmacon Research Foundation, Inc. (Chairman), 1979-

Editorial Board:

Drug Metabolism and Disposition, 1972-
Toxicology and Applied Pharmacology, 1981-1989

University of Kentucky Committees:

College of Medicine Second Year Curriculum (Chairman), 1973-1975
College of Medicine Second Year Promotions, 1973-1975
College of Medicine Educational Policy, 1972-1976
College of Medicine Pharmacy and Therapeutics, 1972-1976
Student Evaluation and Academic Policy for Second Year (Chairman), 1975-1976
Academic Area Advisory Committee for Biological Sciences, 1974-1976 (Chairman, 1975-1976)
Graduate School Fellowships for Biological Sciences, 1974-1976 (Chairman, 1975-1976)
Ad Hoc Committee to Review College of Pharmacy (Chairman), 1976
Ad Hoc Committee to Review Institute of Environmental Sciences, 1976

University of Louisville Committees:

School of Medicine Biomedical Hazards Committee (Chairman), 1977-1983
School of Medicine Insitutional Self-Study Task Force for LCME Accreditation (Chairman), 1979-1980
School of Medicine Radiation Safety Committee (Chairman), 1980-1983

Cancer Center Advisory Committee, 1978-1983
Biological Hazards Committee, 1980-1983

Commonwealth of Kentucky Committees:

Kentucky Drug Formulary, 1977-1981
Kentucky Tobacco and Health Institute Technical Advisory Committee, 1977-1983
Kentucky Medical Assistance Program, 1978-1981
Kentucky Heart Association Research Review Committee, 1980-1981

National Committees:

Guidelines for Detection of Hepatotoxicity Due to Drugs and Chemicals; Fogarty International Center, 1978
Reproductive Effects Assessment Group (Teratology); Environmental Protection Agency, 1980
Educational Affairs Subcommittee of ASPET: New Approaches to Teaching Pharmacology, 1980-1983
ASPET/SOT 1982 Joint meeting in Louisville, Local Committee Chairman
Educational Affairs Committee of ASPET, Chairman, 1983-1986
Society of Toxicology Committee on Information Handling, 1986-1988
FASEB Education Committee, 1986-1989
Texas Coordinating Board, review of teaching programs, 1983, 1985, 1987, 1990

International Committees:

Alfred Benzon Symposium III, Copenhagen and Lund, Ion Homeostasis of the Brain, May, 1970
International Agency for Research on Cancer, Alcohol Drinking, Lyon, France, October, 1987
International Life Sciences Institute, Ethyl Carbamate, Brussels, Belgium, June, 1989

WILLIAM J. WADDELL

Publications

Butler, T.C. and Waddell, W.J.: A pharmacological comparison of the optical isomers of 5-ethyl-5-phenyl hydantoin (Nirvanol) and of 3-methyl-5-ethyl-5-phenyl hydantoin (Mesantoin). *J. Pharmacol. Expt. Ther.* 110:120-125, Jan. 1954.

Butler, T.C. and Waddell, W.J.: The role of the liver in the demethylation of N-methyl derivatives of hydantoin and of 2,4-oxazolidinedione. *J. Pharmacol. Expt. Ther.* 110:241-243, Feb. 1954.

Butler, T.C., Mahaffee, C. and Waddell, W.J.: Phenobarbital: studies of elimination, accumulation, tolerance, and dosage schedules. *J. Pharmacol. Expt. Ther.* 111:425-435, Aug. 1954.

Butler, T.C. and Waddell, W.J.: A pharmacological comparison of the optical isomers of 5-ethyl-5-methyl-2,4-oxazolidinedione and of 3,5-dimethyl-5-ethyl-2,4-oxazolidinedione (Paramethadione, Paradione). *J. Pharmacol. Expt. Ther.* 113:238-240, Feb. 1955.

Waddell, W.J.: Lysis of dog erythrocytes in mildly alkaline isotonic media. *Am. J. Physiol.* 186:339-342, Aug. 1956.

Waddell, W.J.: A simple ultraviolet spectrophotometric method for the determination of protein. *J. Lab. Clin. Med.* 48:311-314, Aug. 1956.

Butler, T.C. and Waddell, W.J.: Metabolic conversion of primidone (Mysoline) to phenobarbital. *Proc. Soc. Expt. Biol. Med.* 93:544-546, Dec. 1956.

Waddell, W.J. and Butler, T.C.: The distribution and excretion of phenobarbital. *J. Clin. Invest.* 36:1217-1226, Aug. 1957.

Butler, T.C. and Waddell, W.J.: Metabolic deethylation of 5,5-dimethyl-3-ethyl-2,4-oxazolidinedione (Dimedion). *Arch. Int. Pharmacodyn. Ther.* 111:308-313, Aug. 1957.

Waddell, W.J. and Butler, T.C.: Renal excretion of 5,5-dimethyl-2,4-oxazolidinedione (product of demethylation of trimethadione). *Proc. Soc. Expt. Biol. Med.* 96:563-565, Dec. 1957.

Butler, TC. and Waddell, W.J.: N-methylated derivatives of barbituric acid, hydantoin, and oxazolidinedione used in the treatment of epilepsy. *Neurology* 8 (Suppl. 1:106-112, April 1958.

Waddell, W.J. and Butler, T. C.: Calculation of intracellular pH from the distribution of 5,5-dimethyl-2,4-oxazolidinedione (DMO). Application to skeletal muscle of the dog. *J. Clin. Invest.* 38:720-729, May 1959.

Butler, T.C. and Waddell, W.J.: The metabolic fate of 5-ethyl-1-methyl-5-phenyl hydantoin (1-methylnirvanol) and that of 3,5-diethyl-5-phenyl hydantoin (3-ethylnirvanol). *J. Pharmacol. Expt. Ther.* 127:171-174, Nov. 1959.

Waddell, W.J. and Hardman, H.F.: Intracellular pH of isolated perfused turtle heart. *Am. J. Physiol.* 199:1112-1114, Dec. 1960.

Waddell, W.J. and Butler, T.C.: Metabolic demethylation of 1,3-dimethyl-5-ethyl-5-phenyl hydantoin (dimethylnirvanol). *J. Pharmacol. Expt. Ther.* 132:291-294, June 1961.

Smith, J.A., Waddell, W.J. and Butler, T.C.: Demethylation of N-methyl derivatives of barbituric acid, hydantoin, and 2,4-oxazolidinedione by rat liver microsomes. *Life Sci.* 2:486-492, July 1963.

Poole, D.T., Butler, T.C. and Waddell, W.J.: Intracellular pH of the Ehrlich ascites tumor cell. *J. Natl. Cancer Inst.* 32:939-946, April 1964.

Butler, T.C., Poole, D.T. and Waddell, W.J.: 5,5-Dimethyl-2,4-oxazolidinedione-2-C^{14} (DMO-C^{14}) and inulin-carboxyl-C^{14} for measurement of intracellular pH. In: Roth, L.J. (Ed.): *Isotopes in Experimental Pharmacology*. Chicago, University of Chicago Press, Chapter 17, pp. 205-210, 1965.

Waddell, W.J.: A simple, rapid method for paper chromatography of barbiturates. *Clin. Chem.* 11:37-39, Jan. 1965.

Chamberlin, H.R., Waddell, W.J. and Butler, T.C.: A study of the product of demethylation of trimethadione in the control of petit mal epilepsy. *Neurology* 15:449-454, May 1965.

Butler, T.C., Waddell, W.J. and Poole, D.T.: Demethylation of trimethadione and metharbital by rat liver microsomal enzymes: substrate concentration-yield relationships and competition between substrates. *Biochem. Pharmacol.* 14:937-942, June 1965.

Waddell, W.J.: The metabolic fate of 5-allyl-5-(1-methylbutyl)barbituric acid (secobarbital). *J. Pharmacol. Expt. Ther.* 149:23-28, July 1965.

Butler, T.C., Kuroiwa, Y., Waddell, W.J. and Poole, D.T.: Effects of 5,5-dimethyl-2,4-oxazolidinedione (DMo) on acid-base and electrolyte equilibria. *J. Pharmacol. Expt. Ther.* 152:62-66, April 1966.

Butler, T.C., Waddell, W.J. and Poole, D.T.: The pH of intracellular water. *Ann. N.Y. Acad. Sci.* 133:73-77, April 1966.

Butler, T.C., Poole, D.T. and Waddell, W.J.: Acid-labile carbon dioxide in muscle: its nature and relationship to intracellular pH. *Proc. Soc. Expt. Biol. Med.* 125:972-974, July 1967.

Butler, T.C, Waddell, W.J. and Poole, D.T.: Intracellular pH based on the distribution of weak electrolytes. *Fed. Proc.* 26:1327-1332, Sept.-Oct. 1967.

Waddell, W.J. and Brinkhous, W.K.: The Ullberg technique of whole-body autoradiography *J. Biol. Phot. Assoc.* 35:147-154, Nov. 1967.

Waddell, W.J.: Distribution of urea-^{14}C in pregnant mice studied by whole-body autoradiography. *J. Appl. Physiol.* 24:828-831, June 1968.

Waddell, W.J., Ullberg, S. and Marlowe, C.: Localization of the bicarbonate and carbonate pools by whole-body autoradiography. *Arch. Int. Physiol. Bioch[e]m.* 77:1-9, Feb. 1969.

Waddell, W.J., and Marlowe, C.: A hydraulic powered microtome in a commercial freezer; an improved cryostat for large sections. *Stain Technol.* 44:81-85, March 1969.

Waddell, W.J. and Bates, R.G.: Intracellular pH. *Physiol. Rev.* 49:285-329, April 1969.

Palmer, K.H., Fowler, M.S., Wall, M.E., Rhodes, L.S., Waddell, W.J. and Baggett, B.: The metabolism of R(+)-and RS-pentobarbital. *J. Pharmacol. Expt. Ther.* 170: 355-363, Dec. 1969.

Waddell, W.J.: Intracellular pH. In: Siesjo, B.K. and Sorensen, S.C. (Eds.): *Alfred Benzon Symposium III: Ion Homestasis of the Brain: The regulation of hydrogen and potassium ion concentrations in cerebral intra- and extracellular fluids.* New York, Academic Press, pp. 233-243, 1971.

Mayberry, H.E., Van den Brande, J.L., Van Wyk, J.J. and Waddell, W.J.: Early localization of ^{125}I-labeled human growth hormone in adrenals and other organs of immature hypophysectomized rats. *Endocrinology* 88: 1309-1317, June 1971.

Waddell, W.J.: Autoradiography in drug distribution studies. In: La Du, B.N., Mandel, H.G., and Way, E.L. (Eds.): *Fundamentals of Drug Metabolism and Drug Disposition.* Baltimore, Williams and Wilkins Co., Chapter 24, pp. 505-514, 1971.

Waddell, W.J.: The distribution of cortisone-^{14}C in pregnant mice. *Teratology* 4:355-365, Aug. 1971.

Hicks, E.P., Cooper, C.W. and Waddell, W.J.: Whole-body autoradiography of the distribution of ^{125}I-thyrocalcitonin in mice. *J. Dent. Res.* 50:1307-1313, Sept.Oct. 1971.

Waddell, W.J. and Wynne, W.P.: The effect of pretreatment with phenobarbital on the teratogenic action of chlorcyclizine in mice. *Arch. Int. Pharmacodyn. Ther.* 194: 168-173, Nov. 1971.

Waddell, W.J.: Localization and metabolism of drugs in the fetus. *Fed. Proc.* 31:52-61, Jan.-Feb. 1972.

Waddell, W.J.: Subcellular and molecular aspects of intracellular pH. *Chest* 61 (Suppl.):56S-59S, Feb. 1972.

Waddell, W.J. and Mirkin, B.L.: Distribution and metabolism of diphenylhydantoin-^{14}C in fetal and maternal tissues of the pregnant mouse. *Biochem. Pharmacol.* 21: 547-552, Feb. 1972.

Waddell, W.J.: The distribution of hydrocortisone-^{14}C corticosterone-^{14}C, and deoxycorticosterone-^{14}C in pregnant mice. *Teratology* 5:219-221, April 1972.

Kennedy, J.S. and Waddell, W.J. Whole-body autoradiography of the pregnant mouse after administration of ^{14}C-^{9}-THC. *Toxicol. Appl. Pharmacol.* 22:252-258, June 1972.

Waddell, W. J.: The role of carbonic anhydrase in the control of intracellular pH. In: Brewer, G.J. (Ed.): *Hemoglobin and Red Cell Structure and Function.* New York, Plenum Press, pp. 215-224, 1972.

Waddell, W.J.: A molecular mechanism for biological calcification. *Biochem. Biophys. Res. Comm.* 49:127-132, Oct. 1972.

Waddell, W.J.: Localization of drugs in tissues with respect to their physiological actions. *Annu. Rev. Pharmacol.* 13:153-168, 1973.

Waddell, W.J.: The distribution of chlorcyclizine-^{14}C in pregnant mice. *Drug Metab. Dispos.* 1:598-601, 1973.

Waddell, W.J. and Baggett, B.: Anesthetic and lethal activity in mice of the stereoisomers of 5-ethyl-5-(1-methylbutyl)barbituric acid (pentobarbital). *Arch. Int. Pharmacodyn. Ther.* 205:40-44, Sept. 1973.

Waddell, W.J., Wilk, A.L., Pratt, R.M., Jr. and Steffek, A.J.: Whole-body autoradiography of the distribution of β-aminopropionitrile in pregnant mice and rat fetuses. *Teratology* 9:211-216, April 1974.

Mayberry, H.E. and Waddell, W.J.: Distribution of ^{125}I-labeled human growth hormone in immature, hypophysectomized rate pretreated with ACTH or unlabeled human growth hormone. *Anat. Rec.* 179:107-114, May 1974.

Waddell, W.J.: Role of membrane-bound enzymes in biological transport. In: Csaky, T.Z. (Ed.): *Intestinal Absorption and Malabsorption.* New York, Raven Press, pp. 37-44, 1975.

Waddell, W.J. and Marlowe, C.: Whole-body autoradiographic studies of the distribution and metabolism of nicotine-^{14}C. *Proc. of the Tobacco and Health Research Institute Symposium on Nicotine and Carbon Monoxide*, pp. 20-32, Nov. 1975.

Waddell, W.J. and Marlowe, G.C.: Disposition of drugs in the fetus. In: Mirkin, B.L. (Ed.): *Perinatal Pharmacology and Therapeutics.* New York, Academic Press, Chapter 3, pp. 119-268, 1976.

Deak, S.T., Ssaky, K.G. and Waddell, W.J.: Localization of histochemical correlation of ^{75}AS by whole-body autoradiography in mice. *J. Toxicol. Environ. Health* 1:981-984, July 1976.

Tucker, A.N. and Waddell, W.J.: Distribution and effect of the weak acids 5,5-dimethyl-2,4-oxazolidinedione (DM0) and 2,4-dinitrophenol (DNP) in membrane vesicles of *micrococcus denitrificans. Arch. Biochem. Biophys.* 176:21-27, Sept. 1976.

Utley, J.F., Marlowe, C. and Waddell, W.J.: Distribution of ^{35}S-labeled WR-2721 in normal and malignant tissues of the mouse. *Radiat. Res.* 68:284-291, Nov. 1976.

Waddell, W.J. and Marlowe, C.: Localization of nicotine-^{14}C, cotinine-^{14}C, and nicotine-l'-N-oxide-^{14}C in tissues of the mouse. *Drug Metabol. Dispos.* 4:530-539, Nov.-Dec. 1976.

Waddell, W.J., Marlowe, C., Miripol, J.E. and Garvin, P.J.: The distribution in mice of intravenously administered [^{14}C]di-2-ethylhexyl phthalate determined by whole-body autoradiography. *Toxicol. Appl. Pharmacol.* 39: 339-353, Feb. 1977.

Lyman, G.E. and Waddell, W.J.: Autoradiography of the water compartments in developing teeth of young mice. *Am. J. Physiol.* 232:F358-F363, April 1977.

Lyman, G.E. and Waddell, W.J.: pH Gradients in the developing teeth of young mice from autoradiography of [^{14}C]DM0. *Am. J. Physiol.* 232:F364-F367, April 1977.

Waddell, W.J. and Marlowe, C.: Autoradiography. In: Garrett, E.R. and Hirtz, J.L. (Eds.): *Drug Fate and Metabolism: Methods and Techniques.* New York, Marcel Dekker, Inc., Vol. 1, Chapter 1, pp. 1-25, 1977.

Waddell, W.J.: Phylogenetic evolution of mechanisms for the termination of drug action. In: B.L. Mirkin, (Ed.): *Clinical Pharmacology.* Chicago, Year Book Medical Publishers, Chapter 2, pp. 23-34, 1978.

Waddell, W.J. and Marlowe, C.: Inhibition by metyrapone of the accumulation of nicotine-^{14}C in bronchial epithelium of mice. *Arch. Int. Pharmacodyn. Ther.* 234: 294-307, Aug. 1978.

Waddell, W.J. and Marlowe, C.: Cortisone mediated decrease in fetal metabolism of glucose. *Arch. Int. Pharmacodyn. Ther.* 242:31-34, Nov. 1979.

Waddell, W.J. and Marlowe, C.: Tissue and cellular disposition of paraquat in mice. *Toxicol. Appl. Pharmacol.* 56:127-140, Oct. 1980.

Waddell, W.J. and Marlowe, C.: Localization of [^{14}C] nitrosonornicotine in tissues of the mouse. *Cancer Res.* 40:3518-3523, Oct. 1980.

Waddell, W.J. and Marlowe, C.: Biochemical regulation of the accessibility of teratogens to the developing embryo. In: Juchau, M.R. (Ed.): *The Biochemical Basis of Chemical Teratogenesis*. New York, Elsevier North-Holland, Chapter 1, pp. 1-62, 1981.

Waddell, W.J. and Marlowe, C.: Transfer of drugs across the placenta. *Pharmacol. Ther.* 14:375-390, 1981.

Liang, Y.-Y., Marlowe, C. and Waddell, W.J.: Autoradiographic study on tissue localization of [^{14}C]cucurbitine in mice. *Acta Pharmacol. Sin.* 3:267-269, December 1982.

Keeney, A.H., Waddell, W.J. and Perraut, T.C.: Carcinogenesis and nicotine in malignant melanoma of the choroid. *Tr. Am. Ophth. Soc.* 80:132-142, 1982.

Marlowe, C. and Waddell, W.J.: Affinity of [^{14}C]nitrosopiperidine and metabolites for mouse epithelial tissues. *Toxicol. Appl. Pharmacol.* 67:110-116, January 1983.

A1-Wathiqui, M.H., Marlowe, C. and Waddell, W.J.: Autoradiographic determination of the effect of total parenteral nutrition on the disposition of ^{14}C-morphine in mice. *Drug Metab. Dispos.* 11:172-174, Mar.-Apr. 1983.

Waddell, W.J. and Marlowe, C.: Inhibition by alcohols of the localization of radioactive nitrosonornicotine in sites of tumor formation. *Science* 221:51-53, July, 1983.

Lachance, M.P., Marlowe, C. and Waddell, W.J.: Autoradiographic disposition of [1-methyl-^{14}C]- and [2-^{14}C] caffeine in mice. *Toxicol. Appl. Pharmacol.* 71:237-241, 1983.

Waddell, W.J.: Whole-body autoradiography for development of new products—evaluation of results. *J. Jap. Cosm Sci. Soc.* 8:135-142, 1984.

Waddell, W.J., Marlowe, C. and Friedman, M.A.: The distribution of [^{14}C]caprolactam in male, female and pregnant mice. *Food Chem. Toxicol.* 4:293-303, 1984.

Egashira, T. and Waddell, W.J.: Histochemical localization of monoamine oxidase in whole-body, freeze-dried sections of mice. *Histochem. J.* 16:919-929, 1984.

Egashira, T. and Waddell, W.J.: Histochemical localization of primary and secondary alcohol dehydrogenases in whole-body, freeze-dried sections of mice. *Histochem. J.* 16:931-940, 1984.

Waddell, W.J., Marlowe, C., and Friedman, M.A.: Autoradiography of ^{14}C-caprolactam. *Proc. Symp.: An Industrial Approach to Chemical Risk Assessment: Caprolactam and Related Compounds as a Case Study.* Pittsburgh, Industrial Health Foundation, pp. 220-228, 1984.

Lachance, M.P., Marlowe, C. and Waddell, W.J.: Autoradiographic disposition of ^{14}C-caffeine in male, female, and pregnant mice. *J. Food Safety* 7:155-160, 1985.

Liang, Y.-Y., Marlowe, C. and Waddell, W.J.: Disposition of [^{14}C]dimercaptosuccinic acid in mice. *Fundam. Appl. Toxicol.* 6:532-540, 1986.

Marlowe, C., Clark, M.J., Mast, R.W., Friedman, M.A. and Waddell, W.J.: The distribution of [^{14}C]acrylamide in male and pregnant Swiss-Webster mice studied by whole-body autoradiography. *Toxicol. Appl. Pharmacol.* 86:457-465, 1986.

Liang, Y.-Y., Marlowe, C. and Waddell, W.J.: Distribution of [nitrile-^{14}C]rorifone in mice by whole body autoradiography. *Acta Pharmacol. Sin.* 8:147-149, 1987.

Waddell, W.J., Marlowe, C. and Pierce, W.M., Jr.: Inhibition of the localization of urethane in mouse tissues by ethanol. *Food Chem. Toxicol.* 25:527-531, 1987.

Yamamoto, T., Pierce, W.M., Jr., Hurst, H.E., Chen, D. and Waddell, W.J.: Inhibition of the metabolism of urethane by ethanol. *Drug Metab. Dispos.* 16: 355-358, 1988.

Waddell, W.J.: Judgments vs. decrees in chemical carcinogenesis. Ethanol: recent IARC decision. The Toxicology Forum, Winter 1988, pp. 172-184.

Waddell, W.J., Marlowe, C., Rao, M.S., and Reddy, J.K.: In vivo distribution of a carcinogenic hepatic peroxisome proliferator: whole-body autoradiography of [^{14}C]ciprofibrate in the mouse. *Carcinogenesis* 10:221-223, 1989.

Waddell, W.J., Marlowe, C., Yamamoto, T. and Clark M.J.: Inhibition of the localization and metabolism of urethane in the mouse by dimethylsulfoxide (DMSO). *Drug Metab. Dispos.* 17:469-472, 1989.

Waddell, W.J., Lech, J.J., Marlowe, C., Kleinow, K.M., and Friedman, M.A.: The distribution of [^{14}C]acrylamide in rainbow trout studied by whole-body autoradiography. *Fundam. Appl. Toxicol.* 14:84-87, 1990.

Yamamoto, T., Pierce, W.M., Jr., Hurst, H.E., Chen, D., and Waddell, W.J.: Ethyl carbamate metabolism: *in vivo* inhibitors and *in vitro* enzymatic systems. *Drug Metab. Dispos.* 18:276-280, 1990.

Kurata, N., Kemper, R., Hurst, H.E., and Waddell, W.J.: Inhibition of the metabolism of ethyl carbamate by acetaldehyde. *Drug Metab. Dispos.* 18:504-507, 1990.

Kurata, N., Hurst, H.E., Kemper, R.A. and Waddell, W.J.: Studies on induction of metabolism of ethyl carbamate in mice by ethanol. *Drug Metab. Dispos.* 19:239-240, 1991.

Kurata, N., Hurst, H.E., Benz, F.W., Kemper, R.A., and Waddell, W.J.: Studies on inhibition and induction of metabolism of ethyl carbamate by acetone and related compounds. *Drug Metab. Disp.* 19:388-393, 1991.

Waddell, W.J., Borzelleca, J.F., Doull, J., Grasso, P., LeBourhis, B., Levy, P.S., and Tamburro, C.H.: Alcohol and Cancer. *Br. J. Cancer* 66:1200-1201, 1992.

Waddell, W.J.: Multiple chemical sensitivity: Fact or fiction? *Health & Environment Digest* 6:5-7, 1993.

Waddell, W.J.: Human risk factors to naturally occurring carcinogens: Thresholds, inhibitors and inducers. *J. Toxicol. Sci.* 18(2) (in press, 1993).

Waddell, W.J.: The science of toxicology and its relevance to MCS. *Regulatory Toxicology and Pharmacology* (in press, 1993).

WILLIAM J. WADDELL

Abstracts, Book Reviews, Notes

Waddell, W.J.; Lysis of dog erythrocytes in mildly alkaline isotonic media. *Fed. Proc.* 15:494-495, Mar. 1956.

Butler, T.C. and Waddell, W.J.: Effects of change of pH on the distribution of phenobarbital in tissues. *J. Pharmacol. Expt. Ther.* 119:137, Feb. 1957.

Waddell, W.J. and Butler, T.C.: Factors affecting the renal elimination of phenobarbital. *J. Pharmacol. Expt. Ther.* 119:191, Feb. 1957.

Waddell, W.J. and Butler, T.C.: The distribution and excretion of 5,5-dimethyl-2,4-oxazolidinedione (the product of demethylation of trimethadione). *J. Pharmacol. Expt. Ther.* 122:79A, Jan. 1958.

Waddell, W.J.: The metabolic conversion of secobarbital to 5-allyl-5-(3-hydroxy-1-methylbutyl) barbituric acid. *Fed. Proc.* 21:182, Mar.-Apr. 1962.

Waddell, W.J.: The metabolic conversion of secobarbital to 5-(1-methylbutyl) barbituric acid and to 5-(2,3-dihydroxypropyl)-5-(1-methylbutyl) barbituric acid. *Fed. Proc.* 22:480, Mar.-Apr. 1963.

Waddell, W.J., Smith, J.A., and Butler, T.C.: The N-demethylation of barbiturates, hydantoins, and trimethadione by rat liver microsome preparations. *Pharmacologist* 5:240, Fall 1963.

Poole, D.T. Butler, T.C., and Waddell, W.J.: Intracellular pH of the Ehrlich ascites tumor cell. *Pharmacologist* 5:270, Fall 1963.

Waddell, W.J. and Marlowe, G.C.: The tissue distribution of 2-C^{14}-5,5-dimethyl-2,4-oxazolidinedione (C^{14}-DMO), C^{14}-inulin, and C^{14}-urea studied with whole body autoradiography. *Proc. 3rd Int. Cong. Nephrol.* II:292, 1966.

Waddell, W.J.: Localization of the bicarbonate and carbonate pools by whole-body autoradiography. *J. Dent. Res.* 47 (Suppl.): 170, Mar. 1968.

Waddell, W.J. and Marlowe, C.: The distribution of cortisone-^{14}C and deoxycorticosterone-^{14}C in pregnant A/JAX mice. *Pharmacologist* 10:155, Fall 1968.

Palmer, K.H., Rhodes, L.S., Fowler, M.S., Waddell, W.J., Baggett, B., and Wall, M.E.: Metabolism of (+)pentobarbital sodium. *Pharmacologist* 10:190, Fall 1968.

Waddell, W.J. and Marlowe, C.: Accumulation of cortisone-^{14}C in the uterine lumen of pregnant A/JAX mice. *J. Dent. Res.* 48 (Suppl.):169, Mar. 1969.

Waddell, W.J. and Marlowe, C.: The distribution of teratogenic agents in pregnant mice. *Teratology* 2:273, Aug. 1969.

Mayberry, H.E., Van den Brande, J.L., Van Wyk, J.J. and Waddell, W.J.: Autoradiographic distribution of ^{125}I-labeled human growth hormone in immature, hypophysectomized rats. *Anat. Rec.* 166:347, Feb. 1970.

Hammarstrom, L., Waddell, W.J. and Hanker, J.S.: Enzymatic activity in ameloblasts at different stages of enamel formation. *J. Dent. Res.* 49 (Suppl.):108, Mar. 1970.

Hicks, E.P., C.W. and Waddell, W.J.: Whole-body autoradiography of the distribution of ^{125}I-thyrocalcitonin in mice. *J. Dent. Res.* 49 (Suppl.):77, Mar. 1970.

Waddell, W.J.: Uses of labeled compounds. *International Conference on Radioactive Isotopes in Pharmacology Science* 168:241, Apr. 10, 1970.

Waddell, W.J. and Mirkin, B.L.: The distribution of diphenylhydantion in pregnant mice. *J. Dent. Res.* 50 (Suppl.):222, Mar. 1971.

Kennedy, J.S. and Waddell[,] W.J.: Wholebody autoradiography of the pregnant mouse after administration of C^{14}-^{9}-tetrahydrocannabinol (^{9}-THC). *Fed. Proc.* 30:279, Mar.-Apr. 1971.

Waddell, W.J. and Steffek, A.J.: The distribution of the lathyrogenic agent, (β-aminopropionitrile-^{14}C (BAPN-^{14}C), in pregnant A/J mice. *Teratology* 4:243-244, May 1971.

Waddell, W.J. and Wynne, W.P.: Reduction in the incidence of cleft palate by phenobarbital in normal and chlorcyclizine treated A/JAX mice. *J. Dent. Res.* 50 (Suppl.):1507-1508, Nov.-Dec. 1971.

Mayberry, H.E. and Waddell, W.J.: Distribution of ^{125}I-HGH in normal and hypophysectomized rats and in hypophysectomized rats pretreated with HGH and ACTH. *Anat. Rec.* 172:366, Feb. 1972.

Waddell, W.J.: A molecular mechanism for biological calcification. *J. Dent. Res.* 51 (Suppl.): 276, Mar. 1972.

Mayberry, H.E. and Waddell, W.J.: Alteration of distribution of ^{125}I-labeled human growth hormone in hypophysectomized rats pretreated with human growth hormone or ACTH. *J. Mitchell Soc.* 88:185-186, Winter 1972.

Waddell, W.J. and Marlowe, G.C.: Whole-body autoradiography of the distribution of nicotine-^{14}C in pregnant mice. *Teratology* 7:A29-A30, June 1973.

Mayberry, H.E., Coffee, J.C., and Waddell, W.J.: Distribution of ^{14}C-estradiol-17-β and ^{14}C-progesterone in pregnant mice. *J. Mitchell Soc.* 89:245, 1973.

Waddell, W.J. and Marlowe, C.: Accumulation of parenterally administered ^{14}C-nicotine in melanin, bronchi and nasal mucosa of mice. *Fed. Proc.* 33:513, Mar. 1974.

Waddell, W14J. and Marlowe, C.: *In vivo* and *in vitro* affinities of nicotine-^{14}C and cotinine-^{14}C for melanin and bronchi of mice. *Fed. Proc.* 34:712, Mar. 1975.

Utley, J.F., Waddell, W.J. and Marlowe, C.: Distribution of ^{35}S-WR2721 in normal and malignant tissues of the mouse. *Radiat. Res.* 62:569-70, 1975.

Waddell, W.J. and Marlowe, C.: Factors affecting the accumulation of ^{14}C-nicotine in bronchial epithelium and melanin. *Fed. Proc.* 35:817, Mar. 1976.

Waddell, W.J. and Marlowe, C.: The distribution in mice of intravenously administered ^{14}C-di-2-ethylhexyl phthalate ([^{14}C]DEHP) determined by whole-body autoradiography. *Toxicol. Appl. Pharmacol.* 37:137, July 1976.

Waddell, W.J. and Marlowe, C.: Teratological significance of drug distributions in the fetus. *Acta Pharmacol. Toxicol.* 41:172-173, May 1977.

Waddell, W.J., Marlowe, C., Miripol, J.E. and Garvin, P.J.: The distribution in pregnant and immature mice of intravenously administered plasma solutions of [^{14}C]di-2-ethylhexyl phthalate determined by whole-body autoradiography. In: Plaa, G.L. and Duncan, W.A.M. (Eds.): *Proc. 1st Int. Cong. Toxicol.* New York, Academic Press, p. 550, 1978.

Waddell, W.J. and Marlowe, C.: Cellular localization of nicotine in bronchial epithelium of mice. *Fed. Proc.* 38: 584, March 1979.

Waddell, W.J., Marlowe, C. and Maling, H.M.: Paraquat disposition in mice by whole-body and cellular autoradiography. *Pharmacologist* 21:212, August 1979.

Waddell, W.J. and Marlowe, C.: Cortisone mediated decrease in fetal metabolism of glucose. *Proc. Second Int. Cong. Toxicol.* Brussels, Belgium, p. 256, 1980.

Waddell, W.J. and Marlowe, C.: Localization of [^{14}C] nitrosonornicotine in tissues of the mouse. *Fed Proc.* 39: 883 March 1980.

Lachance, M.P., Marlowe, C. and Waddell, W.J.: Autoradiographic disposition of ^{14}C-caffeine in mice. *Fed. Proc.* 40:634, March 1981.

Waddell, W.J. and Marlowe, C.: Autoradiographic localization of metabolites of ^{14}C-vinyl chloride in mice. *Toxicologist* 1:78, March 1981.

Waddell, W.J. and Lachance, M.P.: Saccharin and bladder tumors (letter to editor). *Science* 214:134, Oct. 1981.

Waddell, W.J.: This week's citation classic. *Current Contents* 42:21, Oct. 1981.

Waddell, W.J. and Marlowe, C.: Affinity of [^{14}C]nitrosopiperidine for epithelial tissues of the mouse. *Proc. 8th Int. Congress Pharmacol.* Tokyo, Japan, p. 493, 1981.

Lachance, M.P., Marlowe, C. and Waddell, W.J.: Autoradiographic disposition of [8-^{14}C]caffeine in male, female and pregnant mice. *Fed. Proc.* 41:1713, March 1982.

Liang, Y.-Y., Marlowe, C. and Waddell, W.J.: Autoradiographic studies on the tissue localization of [^{14}C] cucurbitine in mice. *Fed. Proc.* 41:5571, March 1982.

Liang, Y.-Y., Marlowe, C. and Waddell, W.J.: Whole-body autoradiographic distribution studies of [^{14}C]dimercaptosuccinic acid in mice. *Pharmacologist* 24:217, August 1982.

Al-Wathiqui, M.H., Marlowe, C. and Waddell, W.J.: Autoradiographic determination of the effect of total parenteral nutrition on the disposition of ^{14}C-morphine in mice. *Pharmacologist* 24:241, August 1982.

Marlowe, C. and Waddell, W.J.: Affinity of [^{14}C]nitrosopyrrolidine and metabolites for mouse epithelial tissues. *Toxicologist* 3:160, March 1983.

Marlowe, C. and Waddell, W.J.: Inhibition by alcohols of the localization of [^{14}C]nitrosonornicotine ([^{14}C]NNN) in epithelial tissues of the mouse. *Fed. Proc.* 42:785, March 1983.

Egashira, T. and Waddell, W.J.: Histochemical localization of MAO activity in whole-body frozen sections of the mouse. *Fed. Proc.* 42:546, March 1983.

Marlowe, C. and Waddell, W.J.[:] Tissue interactions of [^{14}C]retinoic acid in the mouse. *Physiologist* 26:A62, August 1983.

Waddell, W.J. and Marlowe, C.: Whole-body autoradiography of [1-^{14}C] and [2-^{14}C]propionic acid in mice. *Proc. 29th Cong. Int. Union Physiol. Sci.* Sydney, Australia, p. 204, 1983.

Waddell, W.J., Marlowe, C., and Friedman, M.A.: The distribution of [^{14}C]caprolactam in male, female, and pregnant mice. *Proc. 9th Int. Cong. Pharmacol.* London, England, p. 551P, 1984.

Marlowe, C. and Waddell, W.J.: Localization of covalently-bound metabolites of [^{14}C]-nitrosonornicotine in tissues of mice. *Toxicologist* 5:203, 1985.

Mitchell, D.B., Baker, T., Marlowe, C. and Waddell, W.J.: Enhanced liver uptake of liposomes due to incorporation of digalactosylodiglyceride (DGDG) in the lipid bilayer. *Toxicologist* 5:37, 1985.

Marlowe, C., Clark, M.J., Friedman, M.A. and Waddell, W.J.: Disposition of [^{14}C] acrylamide in male and pregnant mice. *Toxicologist* 6:97, 1986.

Waddell, W.J., Lech, J.J., Marlowe[,] C., Kleinow, K.M. and Friedman, M.A.: Whole-body autoradiographic study of [^{14}C]acrylamide in rainbow trout. *Toxicol. Letters* 31 (Suppl.): 129, 1986.

Waddell, W.J. and Marlowe, C.: Maternal-fetal pharmacodynamics. *Proc. Royal Microscopical Soc.* 21:277, 1986.

Waddell, W.J., Marlowe, C. and Pierce, W.M., Jr.: Persistent inhibition of urethane uptake in mouse tissues by ethanol. *Proc. 10th Int. Cong. Pharmacol.*, Sydney, Australia, p. 1198P, 1987.

Tamburro, C.H. and Waddell, W.J.: Letter to the Editor: Cancers of the nasopharynx and oropharynx and formaldehyde exposure. *J. Natl. Cancer Inst.* 79:605, 1987.

Al-Wathiqui, M.H., Hurst, H.E. and Waddell, W.J.: Lowering of blood ethanol concentration in acutely intoxicated rats by continuous infusion of the crystalline amino acid solution, Aminosyn. *Clin. Research* 35:842A, 1987.

Yamamoto, T., Pierce, W.M., Hurst, H.E., Chen, D. and Waddell, W.J.: Inhibition of the metabolism of urethane by ethanol in mice. *Proc. ISSX/SOT Symposium on Endogenous Factors in the Toxicity of Xenobiotics.* Clearwater, Florida, November, 1987.

Long, P.H., Marlowe, C., Custer, D.A., Buring, S.L., Neurbecker, T.A., and Waddell, W.J.: Autoradiographic localization of [^{14}C]EDITEMPA in tissues of the rat. *Proc. 5th Int. Congress on Bone Morphometry*, Nigata, Japan, p. , 1988.

Kurata, N., Kemper, R.A., Hurst, H.E., Pierce, W.M., and Waddell, W.J.: GC/MS determination of urethane pharmacokinetics in mice. *Toxicologist* 9:287, 1989.

Kurata, N., Benz, F.W., Hurst, H.E., Kemper, R.A., and Waddell, W.J.: Inhibition and induction of metabolism of ethyl carbamate (EC) by acetone. *Toxicologist* 10: 189, 1990.

Kurata, N., Hurst, H.E., Kemper, R.A., and Waddell, W.J.: Studies on induction of metabolism of ethyl carbamate (EC) in mice by ethanol. *FASEB J.* 4:A753, 1990.

Kemper, R.A., Kurisaki, E., Hurst, H.E., and Waddell, W.J.: Inhibition and induction of metabolism of ethyl carbamate (EC) in mice: phenobarbital (PB) treatment effects. *Toxicologist* 11:235, 1991.

UNITED STATES DISTRICT COURT
NORTHERN DISTRICT OF GEORGIA
ATLANTA DIVISION

(Title Omitted in Printing)

The deposition of DR. WILLIAM CHARLES BAILEY, taken pursuant to Notice and agreement of counsel and pursuant to the Federal Rules of Civil Procedure and the Federal Rules of Evidence; all formalities waived, excluding the reading and signing of the deposition; before Robin B. Myers, Certified Court Reporter and Notary Public in and for the State of Georgia; commencing at 9:05 a.m., Friday, October 29, 1993 at 1600 The Candler Building, 127 Peachtree Street, Atlanta, Georgia.

* * * *

[4] DR. WILLIAM CHARLES BAILEY
was called as a witness herein and, having been first duly sworn, was examined and deposed as follows:

* * * *

[22] Q. Do you think PCBs could have any effect on the respiratory system if you inhaled it?

A. I know of no documentation or literature that there is, you know, a human cause of this in the human literature.

* * * *

[23] Q. When you say it would not be an unsual thing, is that because physicians often look at animal research to see whether or not there could be a correlation between a given set of circumstances with [24] animals and that same set of circumstances with humans?

A. Well, it's really a little different from that. What is done often is—is animal studies allow one to know

specifically whether or not you want to look in humans for specific things, but then you really can't necessarily correlate animal research to human research until the human research has been done. But clearly, if there would be—it's fairly common in drug studies, for instance, that drugs are used in animals first and if there are any kind of problems in animals, those specific things would be looked at, but—you know, in humans to make sure there wasn't a problem. Most of the time most of the animal research is done at very high doses of things to sort of overwhelm the animal in a sense, and if there's any possible chance of causing disease, then—then it may be seen, and then you begin to try to correlate that to what the human dose might be and see—and look specifically in those areas. But before you can really draw any conclusions about humans, you have to have data on humans, obviously, because they're different.

* * * *

[26] Q. Are you aware of any human health effects associated with dioxin exposure?

[27] A. I'm not aware that dioxin exposure can cause lung cancer or specifically small cell cancer, which was the particular human effect I was interested in in reviewing that literature.

* * * *

[28] Q. Are you familiar with the United States Government's position on dioxins and the association of dioxins in human injury?

A. I'm familiar with a recent report that was produced.

Q. What is your understanding of that report's conclusions concerning lung cancer?

A. I—I actually am confident of what that conclusion is because I just looked at that, and they found really no real evidence of lung cancer and specifically no real evidence of small cell cancer in that exposure.

Q. Which report are you referring to? Do you know the publication that that—

A. The two- —The two-volume set on—that was just published. The chairman of that report was—well, you know, it was just—just published recently, but I—

Q. Within the 90 days?

A. Yeah.

* * * *

[30] Q. Are you aware of any dangers to human health associated with polychlorinated biphenyls?

A. Well, yes.

Q. What are those?

A. It seems to me the—the human data—dangers have been documented. They're primarily some skin problems, chloracne and liver dysfunction.

Q. Any other functions—problems whatsoever?

A. Well, those are the only things that I am familiar that were, you know, have been documented beyond a shadow of a doubt.

Q. Are there any that have been documented not beyond a shadow of a doubt but nevertheless there are some studies supporting claims?

A. Well, of course, I—I basically looked at the literature to draw two conclusions. What were the things that clearly beyond a shadow of a doubt could be caused by PCB, and I think I shared that with you. And then I looked specifically, is there really even any circumstantial evidence that small cell cancer, which is what this man has, could be caused by PCB and found no [31] evidence, even circumstantial or partial or, you know, inconclusive that that might be considered. So, that was kind of, you know, once I went through the literature and came to those conclusions, I really didn't feel like I needed to examine some of the other, less pertinent bits of information.

Q. Well, did you find any information that in any way would have led you to believe that PCBs are a probable human carcinogen?

A. Well, the only data that I saw that's—that's pretty definitive is that it can cause carcinogen in animals, and

so, therefore, you need to be aware, and that's why human studies need to be done, to make sure. But again, I looked specifically for lung cancer and specifically small cell cancer, and no evidence whatsoever that they're—

A. Well, yeah. I—I really don't know the details of—

Q. Well, would you classify it from all the documents that you have read as a probably human carcinogen?

A. Well, I didn't really examine it from that standpoint. I examined it from the standpoint of: Is it likely or even possible that it could have caused small cell cancer, and it's so clear that small cell cancer is caused by cigarette smoking, and there was not even any circumstantial evidence in the literature that it might [32] be related to PCB that I didn't really need to pursue that issue any further.

* * * *

[39] Q. We've just talked about some known dangers of PCBs, and you told me about the skin problems and the liver problems. How about dibenzofurans? Are you aware of any known dangers associated with those chemicals?

A. Well, yeah. I—I really don't know the details of—of these other chemicals and didn't really pursue those in as much, you know, thoroughness, other than looking specifically for the possibility of small cell cancer. And so, the only thing I can say with any confidence to you because it's the only thing I really looked at specifically was that those agents have never been associated with the development of small cell cancer in humans.

Q. How about dioxins?

A. The same thing.

* * * *

[44] Q. [. . .] Why do you think that the table in Plaintiff's Exhibit No. 2 is relevant to your testimony today?

A. Well, one of the things that—that I was looking at is the overwhelming evidence that small cell cancer is exclusively caused by cigarette smoking. And—And so, what we'll do is when we get to the particular table that relates to that is we'll see that, you know, about 99-plus

percent are associated with— [45] you know, documented to be associated with cigarette smoking, and the random misclassification could certain account for that one percent or less.

Q. Okay. What's this next one, No. 3?

A. Let's see. . . This particular table deals with—This particular table deals with various diseases and—from, you know, coronary heart disease, COPD, cancer of the lips, lungs and so forth in both males and females and the attributable risks, taking all cancers or all specific things. This doesn't particularly take out small cell cancer, where one would say almost a hundred percent of small cell cancer would be attributable to—to cigarette smoking. But these would include, for instance, asbestos workers and a number of other people. And the point here is that 90 percent of men, the attributable risk for lung cancer, you know, can be related specifically to cigarette smoking.

* * * *

[48] Q. Plaintiff's Exhibit No. 4.

A. Uh-huh (affirmative), right, okay. This one relates various cell types. As you know, lung cancer can be, you know, a number of different cell types, epidermoid, small cell, adenocarcinoma, large cell and bronchioloalveolar cell carcinoma, five different cell types, and the question here is, you know, what—which of these cell types are related to smoking or more related to smoking and so forth. And you can see here the total numbers, and then you have it in males and females, and so they're—small cell cancer, you have 640 total. This is from the Mayo Clinic, and so these [49] are 640 cases that have gone through the Mayo Clinic. And 533 of those were men that were smokers, and only 4 of the 533 were nonsmokers. And then in women, it's 103.

So, basically, when you—you're talking about really almost—really more than 99 percent of these people were actually smokers and who developed small cell cancer.

Q. Does that chart tell you when the onset of the small cell cancer was, at what age?

A. It doesn't tell when the onset was, it doesn't deal with whether or not they quit smoking or not. It just says that they had been smokers; that's all it says.

Q. Okay.

A. And so, basically, it pretty well documents, and if you think about this first chart that we went over about the possibility of random misclassification, you—these 533 smokers would not have been randomly misclassified, you know, but 4 of the nonsmokers could've been. So, it's conceivable that they were. It's also conceivable, as you say, that—that they may have been—had passive smoking, you know, environmental smoke, as well.

Q. With respect to No. 4 here, that you just had in your hand, —

[50] A. Uh-huh (affirmative).

Q. —does that chart or study show in any fashion whether or not any of the small cell cancer victims had been subjected to any known cancer promoters in addition to their cigarettes?

A. Well, this chart deals strictly with cigarettes, which is basically what we've already seen from the other attributable risk chart; that is, 90 percent of the attributable risks for all cancers, and in this case, it looks like it's 99 to 100 percent of the attributable risk for small cell cancer.

* * * *

Q. Okay. No. 5 here.

A. Okay. No. 5 was basically a printout of one of the questions that—that I was asked was how unusual is it for somebody to be in their 30s when they develop lung cancer, and I said that's relatively unusual, but not unheard of. I mean, I've seen some in their 20s. Personally I've taken care of patients who have developed lung cancer, and so basically, you know, after I was [51] asked that question, I just called our cancer center and asked the statistical people did they have a good printout of all the cancer patients we have. They said, no, they really

didn't, but they could get a few things, like people who had had surgery in the last several years or something like that. And so, this is people that were on the surgical list on the computer in the last five years from the cancer center at UAB. So, these would just be Birmingham patients, under 40.

And actually there is 27 names, but 1 of them, for some reason it looks like it's the same name, Annie B. and Annie Raspberry (ph), so I think it's really just 26 people here over a 5-year period. And as you can see, there's some—some of these cancer patients are 29 and 28, plenty of 30s. And so, like I say, it's—it's unusual, but by no means unheard of, and I see patients like that all the time that do develop cancer.

Q. How many total patients did UAB see for lung cancer?

A. You mean what percentage would this represent?

Q. Yes, sir.

A. I would—I would guess this would represent a relatively small percentage, 10 percent or so, say, of the lung cancers, but I really don't [52] know specifically?

Q. You don't know?

A. No, unh-unh (negative). I would say in my own personal experience that, you know, I personally have seen several people in their 20s that have developed lung cancer, and I've probably seen hundreds of people with lung cancer, and so as far as people in their 20s, I would say that's very rare, you know, what may be less than one percent, just of my own personal experience.

But in the 30s, you know, not all that uncommon.

* * * *

[55] Q. Now, what is No. 6?

A. No. 6, well, that was—This is again an article sort of summarizing all the studies relating to smoking in spouses, of which is among the better—you know, groups of studies with large numbers looking at the possibility of developing lung cancer in nonsmokers. One of the

questions is: Can nonsmokers develop lung cancer, and obviously, a very small percentage do. So, you know, like I say, the 90 percent of attributable risk is to cigarette smoking. And the question is: Within that 10 percent, not just talking about small cell, but all other cancers as well, is there other opportunities to be exposed to cigarette smoke. And clearly, there is if you live in the household with a husband or wife who smoke or you, as you say, go to work and are exposed to somebody or actually grow up in a home as a child. And some of the greater risks really are if the mother smokes for children and if the father smokes and so forth.

But this strictly relates to husbands and wives. And as you can see, the risks, the relative risks in all of these studies range from about two at the lowest to up to nine and—and about seven and—and two are the highest. But most of them are in the two-to-four range, meaning that a nonsmoker in a nonsmoking home has a risk of—of one, say, and that would be the [56] baseline, and if you are a nonsmoker in a smoking home, you have an increased risk of—of developing lung cancer.

Q. And the number there appears on most of the studies to hover around two; is that correct?

A. Yeah, right, two or three. That would be the average, although it goes up to as high as nine in one individual study.

Q. Is two or three—

A. Two or three times the risk.

Q. Two or three times. Can that be statistically significant?

A. Oh, sure, with large enough numbers. And this—this really is a chart that eliminates those studies that don't have large numbers to be statistically significant. So, these are—are studies that are statistically significant. There's probably about 27 or 28 studies from which these—you know that were sufficiently—

Q. What publication is this?

A. This is one of the surgeon general's reports, and this is the front page from that. It's a pretty definitive document.

Q. What is No. —

A. In fact—In fact, all of these— [57] Because I wanted to be sure there was just absolutely reputable sources, all of these come from various surgeon general's reports, and so, there's just not much question about the authenticity of the data.

Q. What's No. 7?

A. One of the other questions that was raised, not only about somebody being relatively young, was that unheard of, the question is: If you were an ex-smoker, did you still have a risk of developing lung cancer, and if you did, what was the risk and when did it fall off and so forth? And so, this is a chart that is develops—develops a model of the relative risk, one being a hundred percent, you know, right after you quit smoking, you know, if you are a smoker. And what happens, —In other words, we all know and we encourage people to quit smoking because there's so many things caused by cigarette smoking that we want to reduce, and this is specifically related to lung cancer. And of course, obviously, it depends—it can depend on whether or not—Smoking duration can make a difference. That can be a variable. It can make a difference in terms of whether it was 10, 20, 30, 40 or 50 years before or not, so this is looking at it both whether you did adjust for smoking duration or whether you did not adjust for smoking duration because that—you know, that would be [58] a variable that could be pertinent.

And as you can see, as I understand it, this particular patient got a varia—varies all over and map in terms of my review of the medical history, but from five to ten years when he quit smoking, I think the average—the average estimate is eight years that he quit smoking, but, you know, it varies from the time he developed lung can-

cer. And anything less than ten years, these two don't diverge much. And as you can see, it gets down to about seventy—above—it's a little above eighty percent, even, in this one at ten years, I think. So, the risk is reducing. And if you don't account for duration, it'll—it'll continue to reduce. But it kind of falls off at about ten years and doesn't reduce a whole lot more if you adjust for duration time.

Q. But in any interpretation, whether you adjust for smoking history or not, any person who stops smoking, —

A. The risk reduces, as you can see.

Q. —at seven years, they've—they've reached a substantial reduced risk in their reduction?

A. It's about—Well, what it is is—If you'll—You'll see this is a hundred percent; that's ninety percent (indicating) of risk, so they've gotten the risk at five years down to about 95 percent.

[59] MR. FLINT: How much?

BY THE WITNESS: (Resuming)

A. Ninety-five percent. So, it's still ninety-five percent of what it was before at five years. So, basically, five years. . . . In fact, I was surprised that it dropped that much the first five years. Most of the time, we don't start counting until after five years, you know. But anyhow, it is at ninety-five percent. It's—It's reduced by five percent at the end of five years.

Q. Okay. So, it's not—

A. In other words, that's not a very substantial reduction in risk.

Q. What is the rate for—

A. It is a statistically significant reduction, however.

Q. Okay. What—

A. It is well worthy of—well worth getting somebody to quit smoking. I mean, you know, no question about that.

* * * *

[61] Q. Would you classify lung cancer as primarily a disease of the older people?

A. Unfortunately, not anymore. It's a disease that we see—As you see, there are, you know, eight thousand—or, you know, four or five thousand deaths a year from people in their 30s or younger. And so. . .

[62] You know, when I first started in this business, basically there were about thirty thousand total cases of lung cancer per year deaths, twenty years ago or something like that, and there are a hundred and fifty thousand deaths per year now, a tremendous increase because of the number of years people have—you know, have been smoking and so forth. And so, it's a—there are—there are a large number of young people who die every year from lung cancer.

Q. When you consider the increase in smoking or increase in lung cancer rates, do you take into account, that in addition to smoking, people's lungs are also assaulted by industrial chemicals on an ever-increasing basis over the same period of time?

* * * *

A. I—You know, I don't know of anything, other than those substances that I mentioned that are associated with that, and those are a pretty specific things. I think asbestos is the main thing that's been found to be a culprit that was in any large number. But [63] we've already looked at it and can see that most of the—all the data would indicate that cigarette smoking is the cause of lung—of small cell cancer, and cigarette smoking is attributable to—is the—is the risk attributable to 90 percent of all cancers of the lung.

* * * *

Q. Well, first, what would you classify it as, primarily an initiator or primarily a promoter or would you say it's a combination of the two or some other answer?

[64] A. Well, the only thing that I know for sure because I don't know that we really understand the mecha-

nism as clearly as would be nice, is that cigarette smoking is the cause of lung cancer in—in basically 90 percent of all cases and in about a hundred percent of small cell cancer. And I don't really know that I can separate it out beyond that. In other words, I think there's a clear etiological relationship. I think that's been documented in the literature, you know, for many, many years, many, many studies, and I think when you get into this—into specifics beyond that I don't that I can really comment intelligently.

* * * *

[68] Q. Do you have an opinion on whether or not PCBs or furans, polychlorinated dibenzofurans, that is, or dioxins are promoters of any cancers in human beings?

A. You know, I don't think that there's any evidence that they're promoters of any cancers, but I know that there's no evidence of lung cancer and of small cell cancer.

Q. Do you know whether or not they've been shown to be promoters of cancer in laboratory animals?

A. I believe there's been an association with the development of cancer in laboratory animals, but the specifics in terms of the mechanism I don't know and have not pursued that. I—I know more that there is an association, and that's—beyond that, I can't give you the specific and technical details of how that association is—or at least what the theories, and I imagine it's really at the theoretical rather than the factual level of that, whether it's a promoter or how it works.

* * * *

[70] Q. Was there anything in Mr. Joiner's history, other than his cigarette smoking which you found in any way significant?

A. As it related to the development of small cell cancer, no, I did not find anything.

* * * *

[74] Q. It's my understanding from the Interrogatory [75] Responses that were provided to us that you were

going to provide us opinions on three things, and I wanted to tell you what those were and see if we've already just talked about them or if we need to go into them more.

The first one is that Mr. Joiner has not suffered any adverse health affects from his alleged exposure to PCBs. And what is your opinion on that?

A. I don't think he has. I don't think the PCB had anything to do with the development of small cell cancer, which is his—his bad health effect.

Q. Can you eliminate as a possibility the idea that Mr. Joiner initiated small cell cancer through his smoking history, but that this small cell cancer was promoted and made to come on at an earlier age because of his exposure to PCBs?

A. There's just no evidence to that effect whatsoever in the literature, and I think his small cell cancer was caused by cigarette smoking.

Q. And it is your opinion, that it could not have been promoted by PCBs?

A. It's my opinion that it was caused by cigarette smoking, and I don't know of any reason to believe that anything else had anything to do with it.

Q. If someone has initiated small cell cancer through cigarette smoking, are you aware of any chemical [76] or product or thing that they could do to promote or increase or speed up the onset of the disease? . . . I want to start with small cell.

A. Well, most of—most of the cancers that are caused by asbestos are adenocarcinoma, the synergistic thing there, but I—I really not rule out the possibility of small cell; I just don't know that for sure with asbestos.

Q. So, it's possible that some chemicals could do it or some products?

A. It—It's conceivable, but basically, if you really look at the statistic on small cell, I have in my personal opinion never seen any case of small cell cancer in anybody but smokers, people who have had cigarette exposure.

And, you know, I have—have seen a number of adenocarcinomas, which is the one that's most often associated with asbestos in nonsmokers, and that's, as you can see in that list, that's the most common thing [77] that you see in nonsmokers. And so, but I just can't speak with any authority because I don't know for sure that that has not been reported. It may have been reported, but, you know, as far as small cell cancer is concerned with asbestos. But that would be the only possibility I can think of.

* * * *

Q. The third thing we were told is that Mr. Joiner's medical complaints are attributable to causes other than exposure to PCBs or products containing PCBs, and from what you've told me today, am I correct in [78] understanding that you contributed—you attribute his lung cancer entirely to his cigarette smoking history?

A. Yes.

Q. And you absolutely do not attribute either the likelihood of the onset of the cancer or the time, that is age, at which the onset occurred to having any kind—to PCBs having been a contributing factor?

A. That's right.

Q. Do you have any other opinions in this case that you've been asked to render?

* * * *

A. Well, I mean, you didn't ask me about that, but I mean, you know, when you're just—when—when you consider the fact that his mother had lung cancer, we talked about genetic predisposition, and I think that is [79] a much more significant factor than anything else if you want to come up with another relationship to the possibility of cause.

Q. At what age did his mother develop cancer?

A. I really don't know.

Q. Do you know what age any other relative that he had developed cancer?

A. No, and again, we're—you know, I mainly responded to your questions I think in the general field of things. That's about all I want to talk about. But if you want to get down to specifics about a little bit more information regarding his risk of lung cancer and his risk of developing small cell cancer, I think—I think we have good data that his family was predisposed to the development of cancer, specifically his mother developing lung cancer and some other uncles and so forth with that problem, as well. He also had two parents that smoked. Both his father and his mother smoked, so he grew up in a home exposed to passive smoking. That's—That's really often associated with the early development of lung cancer. What happens, obviously, is you begin to get the passive smoking risk as an infant, perhaps, and that's continued for many, many years. And what happens is usually people who grow up in that sort of environment begin to smoke earlier because it's just one of those [80] things. It's just a standard—It's part of their environment, and they figure smoking is normal, so they usually begin as early teenagers or, you know, some time in that period of time.

Q. Do you know when Mr. Joiner began?

A. I think he said he was 18, but I mean, I—you know, those are difficult estimates sometimes to know for sure, but it's not unusual for—he was clearly exposed to passive smoking very early, and, you know, the statistical probability would be somebody like that would start sort of early, you know. And so, I think those factors are, you know, additionally important factors in terms of the age at which you develop lung cancer.

* * * *

[81] Q. Do you think there's any need for more studies?

A. Oh, there's always need for more studies to—to document things further, but at this point, I think there's no evidence of any health risk as far as small cell cancer is concerned, you know, in this—this particular exposure.

* * * *

[84] Q. Anything else about his health that you need to know in order to be confident with your opinion or are you as confident as you need to be?

A. Well, related to the questions at hand, yes. The question at hand is, you know, what's the cause of the small cell cancer. I think I've got the data to support that.

* * * *

[Plaintiffs Exhibit 1]

CURRICULUM VITAE

William Charles Bailey, M.D.

Date and Place of Birth:

August 4, 1939
Jacksonville, Florida

Social Security Number:

263-54-0797

Marital status:

Married June 8, 1963;
Wife: Bonnie Shaw Bailey
Children: William C. Bailey, Jr. born January 4, 1965; John Faison-Oates Bailey born April 16, 1969; Evans Cecil Bailey born October 26, 1970

Education:

Washington and Lee University, Lexington, Virginia, 1961, B.A. Tulane University School of Medicine, New Orleans, Louisiana, 1965, M.D.

Internship, Residency and Fellowship:

Rotating Internship, Tulane Division, Charity Hospital, New Orleans, Louisiana, 1965-66.

Residency in Internal Medicine, Tulane Division, Charity Hospital, 1968-70.

National Institute of Health Fellowship, Pulmonary Disease, Tulane University School of Medicine, New Orleans, Louisiana, 1970-1972.

Military:

United States Public Health Service, Surgeon (Lieutenant Commander), assigned as Tuberculosis Control Officer, Jefferson County Health Department, Birmingham, Alabama, 1966-1968.

Previous Appointments:

Director, Bureau of Tuberculosis Control, City of New Orleans Health Department, New Orleans, Louisiana, 1970-1973.

Assistant Clinical Director, Charity Hospital of Louisiana, New Orleans, Louisiana, 1971-1972.

Tuberculosis Control Officer, Charity Hospital of Louisiana, 1971-1973.

Director, Inhalation Therapy, Charity Hospital of Louisiana, 1971-1973.

Director, Respiratory Intensive Care Unit, Charity Hospital of Louisiana, New Orleans, Louisiana, 1971-1973.

Assistant Professor of Medicine, Tulane University School of Medicine, New Orleans, Louisiana, 1972-1973.

Assistant Professor of Medicine, University of Alabama School of Medicine, Birmingham, Alabama, 1973-1975.

Medical Director, Bureau of Communicable Diseases, Jefferson County Health Department, Birmingham, Alabama, 1973-1978.

Associate Professor of Medicine, University of Alabama School of Medicine, Birmingham, Alabama, 1975-1979.

Chief, Pulmonary Disease Section, Veterans Administration Medical Center, Birmingham, Alabama, 1973-1985.

Current Appointments:

Professor of Medicine, University of Alabama School of Medicine, Birmingham, Alabama, 1979-present.

Assistant Dean/Education, University of Alabama at Birmingham, Birmingham, Alabama, 1976-present.

Associate Chief of Staff for Education, Veterans Administration Medical Center, Birmingham, Alabama, 1976-present.

Tuberculosis Coordinator, Alabama Department of Public Health, 1973-present.

Consultant, Bureau of Communicable Diseases, Jefferson County Health Department, Birmingham, Alabama, 1978-present.

Director, Comprehensive Asthma Program, University of Alabama at Birmingham, 1982-present.

Director, Lung Health Center, University of Alabama at Birmingham, 1986-present.

Member, NHLBI Task Force on Research and Education for the Prevention and Control of Respiratory Diseases, June 1988-present.

Member, NHLBI Expert Panel on the Management of Asthma, 1989-present.

Recipient, NHLBI Preventive Pulmonary Academic Award, 1989-1994.

Chairman, NHLBI Task Force on the Prevention of Asthma.

Member, National Heart, Lung, and Blood Advisory Council of the National Institutes of Health, 1991-present.

Member, UAB AIDS Center, 1992-present.

Professional Certifications:

American Board of Internal Medicine—Certification, Internal Medicine, 1972.

American Board of Internal Medicine—Certification, Pulmonary Disease, 1972.

Professional Associations and Committees:

Alabama Public Health Association—Chairman, Section on Tuberculosis, 1976-1977.

Alabama Thoracic Society—President, 1975-1976.

American College of Chest Physicians—Fellow.

American College of Chest Physicians, Steering Committee on Pulmonary Infection, 1976-1979, 1981-1984.

American College of Physicians—Fellow.

American Federation for Clinical Research, 1985.

American Lung Association of Alabama—President, 1987-1989.

American Lung Association of Alabama—Director-at-Large, 1983-1989.

American Medical Association.

American Public Health Association.

American Thoracic Society—Chairman, Tuberculosis Scientific Assembly, 1976-1977.

American Thoracic Society—Planning Committee, 1977-1978.

American Thoracic Society—Council, 1976-1980.

American Thoracic Society—Executive Committee, 1977-1980.

American Thoracic Society—Representative to the International Union Against Tuberculosis, 1981-present.

American Thoracic Society—Chairman, Scientific Assembly on Microbiology, Infectious Disease and Tuberculosis, 1983-1984.

American Thoracic Society—Alabama Representative to the Council of Chapter Representatives, 1982-87.

American Thoracic Society—Board of Directors—1983-1985; 1987-1990.

American Thoracic Society—Chairman, Council of Chapter Representatives, 1986-87.

International Union Against Tuberculosis—Committee on Epidemiology and Statistics, 1979-1986.

International Union Against Tuberculosis—Member, Governing Council, 1982.

International Union Against Tuberculosis—Committee TB Control, 1986-present.

International Union Against Tuberculosis—Secretary of TB Control, 1986 Jefferson County Medical Society.

Jefferson-Shelby Lung Association—Board Member, 1975-1978, 1980-1984.

Medical Association, State of Alabama.

Southern Medical Association—Committee on Publications and Journal Advertising, 1981-1982.

Veterans Administration Central Office Pulmonary Disease Consultant Advisory Committee.

Veterans Administration Pulmonary Physicians' Association—Secretary-Treasurer, 1982-1984.

Veterans Administration Pulmonary Physicians' Association—President-Elect, 1984-1985.

NHLBI Lung Health Study Steering Committee, 1984-present.

Veterans Administration Pulmonary Physicians' Association—President, 1985.

Veterans Administration Southeastern Regional Medical Education Center Program Review Committee.

Veterans Administration Medical Center's Official Representative, AAMC Group on Medical Education, 1987.

UAB Scheduling, Appointments, and Registration Committee, 1990-1991.

UAB Graduate Medical Education Committee, 1990-present.

VA Dean's Committee, 1990-present.

UAB School of Medicine Continuing Medical Education Committee, 1991-present.

Joint Health Sciences Faculty Status Committee, 1991-present.

Centers for Disease Control National Coalition to Eliminate Tuberculosis, ACCP Representative, Steering Committee member, Executive Committee member, 1992-present.

UAB Department of Pastoral Care Advisory Board, 1992-present.

PUBLICATIONS

William C. Bailey, M.D.

1. Bailey, William C.: Discussion of a Portable Device That Allows Emergency Cardiopulmonary Resuscitation to be Performed by One Person. Bulletin of the Tulane Medical Faculty 24:291-296, August 1965.

2. Bailey, William C., Thompson, Doris H., Greenberg, Harry B.: Mycobacterial Infections Among Poor Pregnant Women in New Orleans. The Journal of the Louisiana State Medical Society 124:237-240, July 1972.

3. Bailey, William C., Thompson, Doris H., Greenberg, Harry B.: Indigent Pregnant Women of New Orleans Require Tuberculosis Control Measures. Health Service Reports 87:737-742, October 1972.

4. Ziskind, M., Weill, H., Bailey, W., Jackson, H., and Houser, J.: Special Study of the Effects of Propranalcl and Propractalol on Forced Expiratory Volume and Resting Heart Rate in Normal Subjects. Clinical Pharmacology and Therapeutics 14:137, January 1973.

5. Weill, Hans, Waggenspack, Carmel, Bailey, William, Ziskind, Morton, and Rossiter, Charles: Radiographic and Physiologic Patterns Among Workers Engaged in Manufacture of Asbestos Cement Products: A Preliminary Report. Journal of Occupational Medicine 15:248-252, March 1973.

6. Bailey, William C., Taylor, Sally L., Dascomb, Harry E., Greenberg, Harry B., and Ziskind, Morton M.: Disturbed Hepatic Function during Isoniazid Chemoprophylaxis. American Review of Respiratory Disease 107:523-529, April 1973.

7. Bailey, William C., Taylor, Sally L., Samuels, Monroe S., Dascomb, Harry E., Ziskind, Morton, and DeRouen, Timothy A.: Validity of Serum Glutamic Oxalecetic Transaminase Determinations in Isoniazid Recipients.

American Review of Respiratory Disease 107:670-672, April 1973.

8. Bailey, William C., Thompson, Doris H., Jacobs, Sydney, Ziskind, Morton, and Greenberg, Harry B.: Evaluating the Need for Periodic Recall and Re-Examination of Patients with Inactive Pulmonary Tuberculosis. American Review of Respiratory Disease 107:854-857, May 1973.

9. Altman, Paul, Pumphrey, Del, and Bailey, William C.: Modification to Prevent Deactivation of Bennett Spirometer Alarm System During Mechanical Ventilation. Respiratory Therapy 18:294-295, May-June 1973.

10. LaPointe, Mark, Bailey, William C., and Greenberg, Harry B.: Contact Investigation: An Important Aspect of TB Control. The Journal of the Louisiana State Medical Society 125:197-200, June 1973.

11. Bailey, William C., Thompson, Doris H., Carson, Bonita, and Greenberg, Harry B.: Fatal Hepatic Necrosis in a Woman Receiving Chemoprophylaxis with Isoniazid. The American Journal of Gastroenterology 59: 512-517, June 1973.

12. Bailey, William C., Horwitz, Joni, Brown, Morton, Thompson, Doris H., Ziskind, Morton M., and Greenberg, Harry B.: Use of the Brief MAST Interview to Detect Alcoholics in a Tuberculous Population. Health Services Reports 88:486-488, June-July 1973.

13. Bailey, William C., Greenberg, Harry, and Ziskind, Morton M: Isoniazid Chemoprophylaxis. The Journal of the Louisiana State Medical Society 125:277-278, August 1973.

14. Bailey, William C., and Greenberg, Harry B.: Chest X-Ray Film Not Needed During Antituberculosis Chemoprophylaxis. Journal of the American Medical Association 225:1121-1122, August 27, 1973.

15. Bailey, William C.: Follow-up of Patients with Inactive Tuberculosis. American Review of Respiratory Disease 108:1023-1024, October 1973.

16. Bailey, William C.: Use of New Techniques in Diagnosis of Pulmonary Disease. The Alabama Journal of Medical Sciences 11:107-110, April 1974.

17. Bailey, William C., Weill, Hans, DeRouen, Timothy A., Ziskind, Morton M., Jackson, Henry A., and Greenberg, Harry B.: The Effect of Isoniazid on Transaminase Levels. Annals of Internal Medicine 81:200-202, August 1974.

18. Bailey, William C., Brown, Morton, Buechner, Howard A., Weill, Hans, Ichinose, Herbert, and Ziskind, Morton: Silico-mycobacterial Disease in Sandblasters. American Review of Respiratory Disease 110:115-125, August 1974.

19. Bailey, William C., Thompson, Doris H., and Greenberg, Harry B.: A Technique for Standardizing the Jet Injector and Mantoux Tuberculin Skin Tests. Public Health Reports 89:465-467, September-October 1974.

20. Bailey, William C., DeRouen, Timothy A., Ziskind, Morton M., and Greenberg, Harry B.: Autoanalytic (Colormetric) Determinations of SGOT in Isoniazid Recipients are Reliable. American Review of Respiratory Disease 111:237-238, February 1975.

21. Bailey, W.C., DeRouen, T.A., Ziskind, M.M. and Greenberg, H.B.: Autoanalytic (Colormetric) Determinations of SGOT in Isoniazid Recipients are Reliable. Medikon IV: 5-6, June 10, 1975.

22. Bailey, William C., Bancroft, William H., Jr., and Goetter, William E.: Teaching Blood Gases: An Automated Display. Journal of Medical Education 51:598-599, July 1976.

23. Sheehy, Thomas W., and Bailey, William C.: Alcoholism and Tuberculosis. The Alabama Journal of Medical Sciences 13:392-395, October 1976.

24. Maetz, H. Michael, Sellers, Clyde A., Bailey, William C., and Hardy, George E., Jr.: Lymphocytic Choriomeningitis from Pet Hamster Exposure: A Local Public Health Experience. American Journal of Public Health 66:1082-1085, November 1976.

25. Bailey, William C., Raleigh, James W., and Turner, J.A. Peter: Treatment of Mycobaterial Disease: An Official Statement of the American Thoracic Society. American Review of Respiratory Disease 115:185-187, January 1977.

26. Bailey, William C.: Respiratory Function Evaluation. The Alabama Journal of Medical Sciences 14:43-47, January 1977.

27. Sheehy, Thomas W., Spencer, J. Terrell, and Bailey, William C.: Postgastrectomy Tuberculosis. The Alabama Journal of Medical Sciences 14:158-161, April 1977.

28. Bailey, William C., Sellers, Clyde A., Lantz, Samuel O., and Hardy, George E., Jr.: Chest Radiographs During Isoniazid Therapy. American Review of Respiratory Disease 115:877-878, May 1977.

29. Burk, John R., Bahar, David, Wolf, Frederick S., Greene, Joe, and Bailey, William C.: Nursery Exposure of 528 Newborns to a Nurse with Pulmonary Tuberculosis. Southern Medical Journal 71:7-10, January 1978.

30. Bailey, William C., Sellers, Clyde A., Sutton, Frank D., Jr., Sheehy, Thomas W., and Maetz, H. Michael: Tuberculosis and Alcoholism. A Partial Solution through Detection. Chest 73:183-185, February 1978.

31. Bailey, William C.: Silicosis. In: *Current Therapy, 1978*. Conn, Howard F., ed. Philadelhpia, W. B. Saunders Company, 1978, pp. 142-144.

32. Matez, H. Michael, Pate, Paul, Sellers, Clyde, Bailey, William C., Holmes, Richard, and Hardy, George E., Jr.: Epidemiology and Control of St. Louis Encephalitis in Birmingham, Alabama, 1975. American Journal of Public Health 68(6):588-590, June 1978.

33. Alexander, W. James, and Bailey, William C.: Mycobacterium Avium-Intracellulare from a Vietnamese Refuge. Southern Medical Journal 71(6):733-735, June 1978.

34. Bailey, William C., and Bancroft, William H., Jr.: Blood Gases Taught by Automated Display. Continuing Education 9:70-77, October 1978.

35. Bailey, William C., Raleigh, James W., and Turner, J.A. Peter: Treatment of Mycobacterial Disease. Continuing Education 10:65-71, March 1979.

36. Alexander, W. James, Avent, C. Kirk, and Bailey, William C.: Simple Primary Tuberculosis in an Elderly Woman. Journal of the American Geriatrics Society 27(3):123-125, March 1979.

37. Rose, C.E., Jr., Zerbe, G.O., Lantz, S.O., and Bailey, W.C.: Establishing Priority During Investigation of Tuberculosis Contacts. American Review of Respiratory Disease 119:603-609, April 1979.

38. Hodgkin, M.M., Eidus, L., and Bailey, W.C.: Isoniazid Phenotyping of Black as well as White Patients. Canadian Journal of Physiology and Pharmacology 57:760-763, June 1979.

39. Shin, Myung Soo, Bailey, William C., McReynolds, Richard A., and Shelton, R. Waid, Jr.: Pneumonic Sarcoidosis. Journal of the Medical Association of the State of Alabama, pp. 21-40, November 1979.

40. Bailey, William C.: Diagnosis of Tuberculosis. Clinics in Chest Medicine 1(2):209-217, May 1980.

41. Dickinson, Douglas S., Bailey, William C., Hirschowitz, Basil I., Soong, Seng-Jaw, Eidus, Leslie, and Hodg-

kin, Mary M.: Risk Factors for Isoniazid(INH)-Induced Liver Dysfunction. Journal of Clinical Gastroenterology 3:271-279, September 1981.

42. Where Do We Go From Here? American Lung Association Bulletin, pp. 21-23, March 1982.

43. Simmons, Steven M., Camp, R. Bradford, Mungall, Dennis R., and Bailey, William C.: The Use of Theophylline Clearance to Design an Oral Aminophylline Regimen. The Alabama Journal of Medical Sciences 19(2):142-146, April 1982.

44. Hon. Jeremy K., Hamner, Ronald W., Bailey, William C., and Sheehy, Thomas W.: Metastatic Melanoma Complicated by Disseminated SCG. Southern Medical Journal 75(10):1263-1264, October 1982.

45. Bailey, William C.: Colleagues and patients: Our Unique Obligation. Christian Medical Society Journal 13(4):24-27, 1982.

46. Bailey, William C.: Short-Term Chemotherapy of Tuberculosis. The Alabama Journal of Medical Sciences 20(1):17-18, 1983.

47. Bailey, William C.: Silicosis. In: *Current Therapy, 1983*. Conn, Howard F., ed. Philadelphia, W. B. Saunders Company, 1983, pp. 126-128.

48. Bailey, William C.: *Koch Centennial Memorial*, Garoth Green, Thomas M. Daniel, and Wilmont C. Ball, eds. Chest 83(4):50, April 1983 (book review).

49. Bailey, William C., Albert, Richard K., Davidson, Paul T., Farer, Laurence S., Glassroth, Jeffrey, Kendig, Edwin, Jr., Loudon, Robert G., and Inselman, Laura S.: Treatment of Tuberculosis and other Mycobacterial Diseases. An Official Statement of the American Thoracic Society. American Review of Respiratory Disease 127:790-796, June 1983.

50. Bailey, William C.: Treatment of Tuberculosis. Medical Times III(6):16-22, June 1983.

51. Bailey, William C.: Medical Information Service via Telephone: Respiratory Failure from Obstructive Airways Disease. The Alabama Journal of Medical Sciences 20(3):354-355, July 1983.

52. Farer, Laurence S., Flynn, James P., Reza, Robert J., Bailey, William C., and Albert, Richard K.: Control of Tuberculosis. An Official Statement of the American Thoracic Society. American Review of Respiratory Disease 128:336-342, August 1983.

53. Bailey, William C.: Treatment of Atypical Mycobacterial Disease. Chest 84:625-628, November 1983.

54. Bailey, William C.: Chemotherapy of Tuberculosis. In: *Tuberculosis*. Praeger Monographs in Infectious Disease, Volume 2. Schlossberg, David, ed. New York, Praeger Publishers, 1983, pp. 65-87.

55. Shin, Myung S., and Bailey, William C.: Computed Tomography of Invasive Pleural Mesothelioma. The Journal of Computed Tomography 7:389-394, 1983.

56. Bailey, William C., and Bass, John B., Jr.: Treatment of Tuberculosis and other Mycobacterial Diseases. American Review of Respiratory Disease 129:511, March 1984 (Letter to the Editor).

57. Bailey, William C.: Clinically-derived Mycobacterial Classifications (Letter to the Editor). Chest 85:714-715, 1984.

58. Bailey, William C., Bass, John B., Jr., Hawkins, Jean E. Kubica, George P., and Wallace, Richard J., Jr.: Drug Susceptibility Testing for Mycobacteria. American Thoracic News 10(1):9-10, Winter, 1984.

59. Bailey, William C. (ed.): Clinics in Chest Medicine. Symposium on Asthma:5(4), December 1985.

60. Bailey, William C., Byrd, Richard B., Glassroth, Jeffrey L., Hopewell, Philip C., Reichman, Lee B.: Preventive Treatment of Tuberculosis. Chest 87(2):128S-132S, February 1985.

61. Shin, Myung S., and Bailey, William C.: Alveolar Cell Carcinoma: Diagnostic Pitfalls in Evaluating the Chest Roentgenogram. Southern Medical Journal 78(2):193-195, February, 1985.

62. Bailey, William C.: Smoking Cessation. Chest 88(3):322-323, September 1985.

63. Bailey, William C.: Smoking Cessation. Southern Medical Journal 79(2):223-226, February 1986.

64. Veal, Curtis F., Jr., M.D., and Bailey, William C., M.D.: Mycobacterium Kansasii Infection. In: *Current Therapy of Respiratory Disease-2*. Reuben M. Cherniack, M.D., ed. B.C. Decker, Inc., Publisher, 1986, pp. 94-96.

65. Manzella, B., Windsor, R.A., Bailey, W.C., et al: Winning Against Asthma: An Adult's Self Help Control Guide. University of Alabama at Birmingham, Publisher, pp. 1-53, 1986.

66. Hopewell, P., Bailey, W.C., Cohen, M.L., et al: Supplement: Future Research in TB. Improving Treatment for Tuberculosis. American Review of Respiratory Disease 134(2):417-420, August 1986.

67. Harding, S.M., and Bailey, W.C.: Tuberculosis: 1986. Hospital Medicine 22(9):147-166, September 1986.

68. Heimburger, D.C., Krumdieck, C.L., Alexander, C.B., Birch, R., Dill, S.R., and Bailey, W.C: Localized Folic Acid Deficiency and Bronchial Metaplasia in Smokers: Hypothesis and Preliminary Report. Nutr Int 3(1):54-60, 1987.

69. Veal, C.F., Jr., M.D., and Bailey, W.C., M.D.: Mycobacterium Kansasii Infection. In: *Current Therapy in Internal Medicine-2*. Theodore M. Bayless, M.D., Michael C. Brian, M.D., and Reuben M. Cherniack, M.D., eds., B.C. Decker, Inc., Publisher, 1987, pp. 803-805.

70. Bailey, William C., Keens, Thomas G., Tinstman, Thomas C.: Plotting a Course for Asthma Therapy. Patient Care 21:62-82, May 15, 1987.

71. Bailey, William C., M.D., Richards, James M., Jr., Ph.D., Manzella, Bryn A., M.P.H, et al: Promoting Self-Management in Adults with Asthma: An Overview of the UAB Program Health Education Quarterly 14(3):345-355, Fall, 1987.

72. Bailey, William C., M.D.: State of the Art on Control: Methods and Techniques in 1987, pp. 6-7. In: Proceedings of the Julia M. Jones Preventive Medicine Day—Tuberculosis, in New York City, October 5, 1987, sponsored by the New York Lung Association.

73. Harding, Susan M., and Bailey, William C.: Chemotherapy of Tuberculosis. In: *Clinical Topics in Infectious Disease: Tuberculosis*, David Schlossberg, ed., Springer-Verlag, Publisher, 1988, pp. 39-53.

74. Young, Keith, M.D., and Bailey, William, M.D.: Nontuberculous Mycobacterial Infections: A Rapidly Increasing Danger. The Journal of Respiratory Diseases 9(2):20-34, February 1988.

75. Heimburger, Douglas C., M.D., Alexander, C. Bruce, M.D., Birch, Robert, Ph.D., Butterworth, C.E., Jr., M.D. Bailey, William C., M.D., and Krumdieck, Carlos L., Ph.D., M.D.: Improvement in Bronchial Squamous Metaplasia in Smokers Treated with Folate and Vitamin B12. The Journal of the American Medical Association 259(10):1525-1530, March 11, 1988.

76. Sanders, Colleen, M.D., Nath, J. Hrudaya, M.D., and Bailey, William C., M.D.: Detection of Emphysema with Computed Tomography. Investigative Radiology 23:262-266, April 1988.

77. Bailey, William C.: All Patients Should Receive Directly Observed Therapy in Tuberculosis (Con), pp. 1075-1076. In: Summer, W.R., Jenkinson, S.G., et al:

Controversies in Pulmonary Medicine. American Review of Respiratory Disease 138:1070-1077, October 1988.

78. Bailey, William C., M.D.: Treating Asthma with Drugs. Respiratory World 1(12):3-11, December 1988.

79. Richards, James M., Ph.D., Bailey, William C., M.D., et al: Some Simple Scales for Use in Asthma Research. Journal of Asthma 25(6):363-371, 1988.

80. Young, Keith P., M.D., and Bailey, William C., M.D.: Tuberculosis and Nontuberculous Mycobacterial Diseases. In: *1989 Conn's Current Therapy*, Robert E. Rakel, M.D., ed., W.B. Saunders Company, 1989, pp. 171-175.

81. Brooks, C. Michael Brooks, Ed.D., Richards, James M., Jr., Ph.D., Bailey, William C., M.D., Martin, Beverly, B.S., R.N., Windsor, Richard A., Ph.D., M.P.H., and Soong, Seng-Jaw, Ph.D.: Subjective Symptomatology of Asthma in an Outpatient Population Psychosomatic Medicine 51:102-108, 1989.

82. Richards, Jr., James M. Dolce, Jeffrey J., Windsor, Richard A., Bailey, William C., Brooks, C. Michael, and Soong, Seng-jaw: Patient Characterisetics Relevant to Effective Self-Management: Scales for Assessing Attitudes of Adults Toward Asthma. Journal of Asthma 26(2):99-108, 1989.

83. Bailey, William C., and Manzella, Bryn A.: *Learn Asthma Control in Seven Days*. Board of Trustees of the University of Alabama for the University of Alabama at Birmingham, November 1989.

84. Manzella, Bryn, Brooks, C. Michael, Richards, Jr., James M., Goetter, William E., Windsor, Richard, Bailey, William C., and Soong, Seng-jaw: Assessing the Use of Metered Dose Inhalers by Adults with Asthma. Journal of Asthma 26(4):223-230, 1989.

85. Dunlap, Nancy E., M.D., and Bailey, William C.: Corticosteroids in Asthma. Southern Medical Journal 83(4):428-432, April 1990.

86. Bailey, William C., and Manzella, Bryn A.: *Controlling Asthma: A Brief Guide*. Board of Directors of the University of Alabama for the University of Alabama at Birmingham, May 1990.

87. Glassroth, Jeffrey, Bailey, William C., Hopewell, Philip C., Schecter, Giesella, and Harden, James W.: Why Tuberculosis is Not Prevented. American Review of Respiratory Disease 141:1236-1240, June 1990.

88. Bailey, William C., Richards, James M., Jr., Brooks, C. Michael, Soong, Seng-jaw, Windsor, Richard A., Manzella, Bryn A.: A Randomized Trial to Improve Self-Management Practices of Adults with Asthma. Archives of Internal Medicine 150:1664-1668, August 1990.

89. Reeder, Kenneth P., Dolce, Jeffrey J., Duke, Linda, Raczynski, James M., and Bailey, William C.: Peak Flow Meters: Are They Monitoring Tools or Training Devices? Journal of Asthma 27(4):219-227, August 1990.

90. Bailey, William C., Richards, James M., Jr., Manzella, Bryn A., Brooks, C. Michael, Windsor, Richard A., and Soong, Seng-jaw: Characteristics & Correlates of Asthma in a University Clinic Population. Chest 98(4):821-828, October 1990.

91. Windsor, Richards A., Bailey, William C., Richards, Jr., James M., Manzella, Brynda, Soong, Seng-Jaw, and Brooks, C. Michael: The Evaluation of the Efficacy and Cost Effectiveness of Health Education Methods to Increase Medication Adherence Among Adults with Asthma. American Journal of Public Health 80(12):1-3, December 1990.

92. Dunlap, N.E., Bailey, W.C.: Corticosteroids in Asthma. Clinical Digest Series 1990.

93. Dolce, Jeffrey J., Crisp, Carol, Manzella, Bryn, Richards, James M., Hardin, J. Michael, Bailey, William C.: Medication Adherence Patterns in Chronic Obstructive Pulmonary Disease. Chest 99:837-841, April 1991.

94. *Guidelines for the Diagnosis and Management of Asthma.* Expert Panel Report, National Asthma Education Program, National Heart, Lung, and Blood Institute (1991), June 1991.

95. *Executive Summary: Guidelines for the Diagnosis and Management of Asthma. Expert Panel Report, National Asthma Education Program, National Heart, Lung, and Blood Institute* (1991), June 1991.

96. Sheffer, Albert L., Bailey, William C., Bleecker, Eugene R., et al: Guidelines for the Diagnosis and Management of Asthma. Pediatric Asthma Allergy & Immunology 5:xxi-188, Summer 1991.

97. Groves, Robert H., Bailey, William C., and Buchalter, Scott: Long-term Oxygen Therapy. Chest 100:544-549, August 1991.

98. Groves, Robert H., Jr. and Bailey, William C.: Early- and Late-Phase Asthmatic Response. Clinical Advances in the Treatment of Asthma 2(4):4-7, September 1991.

99. Harding, Susan M. and Bailey, William C.: Update on Tuberculosis. Hospital Medicine 27:21-34, November 1991.

100. Dolce, Jeffrey J., Ph.D., Kohler, Connie L., Dr.P.H., Manzella, Bryn A., M.P.H., and Bailey, William C., M.D.: *Living with COPD: Managing Your Medicines.* The Board of Trustees of The University of Alabama for The University of Alabama at Birmingham, 1991.

101. Bailey, William C., Richards, James M., Brooks, C. Michael, Soong, Seng-jaw, Brannen, A. Lynne: Features of Asthma in Older Adults. Journal of Asthma 29(1):21-28, 1992.

102. Bailey, William C., Higgins, Darlene M., Richards, Bonnie M., Richards, James M., Jr.: Asthma Severity: A Factor Analytic Investigation. The American Journal of Medicine 93(3):263-269, September 1992.

103. Bailey, William C., Clark, Noreen M., Gotsch, Audrey R., Lemen, Richard J., O'Connor, George T., and Rosenstock, Irwin M.: Asthma Prevention. Chest 102(3):216S-231S, September 1992 Supplement.

ABSTRACTS

1. Brown, M., Buechner, H.A., Bailey, W.C., and Ziskind, M.M.: Atypical Mycobacterial Pulmonary Disease at the New Orleans Veterans Hospital and Metropolitan New Orleans. American Review of Respiratory Disease 103:885-886, June 1971.

2. Bailey, W.C., Brown, M., Buechner, H.A., Weill, H., Ziskind, M.: Silico-Mycobacterial Disease in Sandblasters. Transactions of the 31st VA Armed Forces Pulmonary Disease Research Conference IB 15-9:4, January 1972.

3. Bailey, W.C., Taylor, S.L., Dascomb, H.E., Greenberg, H.B., and Ziskind, M.M.: Disturbed Liver Function During INH Chemoprophylaxis. American Review of Respiratory Disease 105:1020, June 1972.

4. Bailey, William C., Brown, Morton, Buechner, Howard A., Weill, Hans, Ichinose, Herbert, and Ziskind, Morton: Silicomycobacterial Disease in Sandblasters. Chest 64:387-388, September 1973.

5. Siskind, Morton M., Weill, Hans, Bailey, William C., Buechner, Howard A., Brown, Morton, Waggenspack, Carmel, Samini, Behzad: Accelerated Silicosis in Sandblasters. Chest 64:411, September 1973.

6. Bailey, William C., DeRouen, Timothy A., Ziskind, Morton M., and Greenberg, Harry B.: Autoanalytic (Colormetric) Determinations of SGOT in Isoniazid Recipients are Reliable. American Review of Respiratory Disease 109:717, June 1974.

7. Bailey, William C., Sellers, Clyde A., and Hardy, George E., Jr.: Once is Enough: Chest Radiographs for Isoniazid Recipients. American Review of Respiratory Disease 111:901, June 1975.

8. Doekel, Robert C., Giesler, Garnett J., and Bailey, William C.: Management of Loculated, Pleural Effusions

and Empyema with Echopleurography. American Review of Respiratory Disease 111:934, June 1975.

9. Bailey, W.C., and Doekel, R.C.: Management of Loculated Pleural Effusions and Empyema with Echopleurography. Medical Instrumentation 10:68, January-February 1976.

10. Sheehy, T.W., Spencer, J.T., and Bailey, W.C.: Post-Gastrectomy Tuberculosis. Clinical Research 389A, April 1976.

11. Dickinson, D.S., Bailey, W.C., and Hirschowitz, B.I.: The Effect of Acetylation Status on Isoniazid (INH) Hepatitis. American Review of Respiratory Disease 115: 395, April 1977.

12. Rose, C.E., Jr., Lantz, S.O., and Bailey, W.C.: Priorities in Investigating Contacts to Active Tuberculosis. American Review of Respiratory Disease 115:406, April 1977.

13. Rose, C.E., and Bailey, W.C.: Supervised Intermittent Chemotherapy of Tuberculosis. American Review of Respiratory Disease 115:407, April 1977.

14. Bailey, W.C., and Sellers, C.A.: Tuberculosis and Alcoholism, Identifying the Problem and Dealing with It Early. Proceedings of the 23rd International Tuberculosis Conference, Mexico City, Mexico, September 22-26, 1975.

15. Dickinson, D.S., Hirschowitz, B.I., Bailey, W.C., Gibson, R., and Hodgkin, M.M.: The Effect of Acetylation (AC) Status on Cholestasis and Hepatocellular Damage (HCD) in Isoniazid (INH) Induced Liver Disease. Gastroenterology 72:1049, Pt. 2, May 1977.

16. Bailey, W.C., and Sutton, F.: Supervised Short-Term Weekly Chemotherapy of Pulmonary Tuberculosis. American Review of Respiratory Disease 117:417, April 1978.

17. Bailey, William C., Sutton, Frank, and Eidus, Leslie: Supervised Short-Term Weekly Chemotherapy of Pulmon-

ary Tuberculosis. Proceedings of the XXIVth World Conference of the IUAT-2, Brussels, Belgium, September 5-9, 1978. Bulletin of the International Union Against Tuberculosis 54(1):12, March 1979.

18. Bailey, William C., Dickinson, Douglas S., Hirschowitz, Basil I., and Hodgkin, Mary M.: Isoniazid Acetylation and Hepatitis. Proceedings of the XXIVth World Conference of the IUAT-2, Brussels, Belgium, September 5-9, 1978. Bulletin of the International Union Against Tuberculosis 54(1):47, March 1979.

19. Bailey, W.C., Sutton, F.D., Jr., and Eidus, L.: The Use of Matrix Isoniazid (INH) in Supervised Short-Term Chemotherapy. American Review of Respiratory Disease 119:394, April 1979.

20. Bailey, W.C., Soong, S.J., and Hirschowitz, B.I.: INH Preventive Therapy—A New Risk: Benefit Analysis. American Review of Respiratory Disease 121:450, April 1980.

21. Bailey, William C., Briggs, Dick D., Jr., and Sutton, Frank D., Jr.: Once a Week Short-Term Tuberculosis Therapy. Program of the XXVth World Conference of the IUAT, Buenos Aires, Argentina, April 21-24, 1982. Bulletin of the International Union Against Tuberculosis 57(1):22, March 1982.

22. Heimburger, D.C., Alexander, C.B., Birch, R., Combs, B., Soto, P., Bailey, W.C., Saxon D., Schnaper, H., and Krumdieck, C.L.: Lower Folic Acid Levels Associated with Bronchial Metaplasia in Smokers. Proceedings of American Association for Cancer Research 26:498, March 1985.

23. Heimburger, D.C., Bailey, W.C., Alexander, C.B., Birch, R., Combs, B., Soto, P., Sacon, D., Schnaper, H., and Krumdieck, C.L.: Bronchial Metaplasia in Smokers Associated with Decreased Folic Acid Levels. American Review of Respiratory Disease 131:A392, April 1985.

24. Heimburger, D.C., Alexander, C.B., Birch, R., Combs, B., Soto, P., Bailey, W.C., Saxon, D., Schnaper, H., and Krumdieck, C.L.: Smokers with Bronchial Metaplasia Have Lower Circulating Folate Levels. Clinical Research 33(2):705A, April, 1985.

25. Glassroth, Jr., Sherman, J., Bailey, W., Alexander, J.W., Hopewell, P., Schecter, G.: Where Does the Tuberculosis Prevention Therapy System Fail? American Review of Respiratory Disease 133(4):A82, April 1986.

26. Bailey, William C., Soong, Seng-jaw, Brooks, C. Michael, et al: Demographics, Co-Morbidity and Medication in an Asthma Population. Chest 92(2):122S, August, 1987.

27. Bailey, William C., Richards, James M., Jr., Manzella, Bryn A., Windsor, Richard A., Brooks, C. Michael, Soong, Seng-jaw: Improving Self-Management Skills of Adults with Asthma. American Review of Respiratory Disease Annual Meeting Supplement 139(4), Part 2:A144, April 1989.

28. Dolce, J.J., Crisp, C., Manzella, B., Bailey, W.C.: Medication Pattern Among COPD Patients in a U.S. Outpatient Clinic. American Review of Respiratory Disease Annual Meeting Supplement 139(4), Part 2:A517, April 1989.

29. Richards, J.M., Jr., Bailey, W.C., Brooks, C.M., Windsor, R.A.: Evaluation of an Intervention for Promoting Self-Management of Adult Asthma. American Psychological Society 1989 Program Summary, pp. 21, June 1989.

30. Richards, B., Bailey, W.C., Brooks, C.M., Windsor, R.A., Soong, S.J., and Richards, J. M.: Judging Symptom Severity of Asthma in a Factor Analytic Study. Clinical Research 38:2, April 1990, pp. 515A.

31. Maisiak, R.S., Brooks, C.M., Richards, J.M., Bailey, W.C.: Racial Differences and Asthma Death: An Analy-

sis of Death Certificates. Clinical Research 38:2, April 1990, pp. 512A.

32. Maisiak, R.S., Richards, J.M., Brooks, C.M., Windsor, R.A., Soong, S.J., and Bailey, W.C.: Racial Differences in the Clinical Characteristics of Asthma Patients. Clinical Research 38:2, April 1990, pp. 512A.

33. Bailey, W.C., Higgins, D.M., Richards, B.M., Richards, J.M.: Asthma Severity: A Factor Analysis. American Review of Respiratory Disease, April 1991 Supplement, pp. A25.

34. Richards, J.M., Brooks, C.M., Kohler, C.L., Martin, B.A., Soong, S.J., Windsor, R.A., Bailey, W.C.: Measuring Adult Adherence to Asthma Medication and Inhaler Regimens: Self-Report Scales. First National Conference on Asthma Management Conference Program, October 11, 1992, Arlington, Virginia, pp. 117.

AUDIOVISUAL PRODUCTIONS

1. Bailey, William C.: Understanding Tuberculosis Today (videorecording). Florida Atlanta University Television, 1967.

2. Briggs, D.D., Jr., Murray, F.J., Bailey, W.C., Bradley, J.D., and Sutton, F.D.: Recent Advances in Pulmonary Diseases. Audio Digest/Internal Medicine 22(12), July 1975.

3. Bailey, William C., and Vath, Richard R.: Asthma: Recent Advances in Diagnosis and Treatment (videorecording), ¾″ u-matic, 58:28 minutes, color. Dental Television, University of Alabama in Birmingham, 1982.

4. Bailey, William C., and Vath, Richard R.: Lung Cancer (videorecording), ¾″ u-matic, 60:27 minutes, color. Dental Television, University of Alabama in Birmingham, 1983.

5. Weill, Hans, Brigham, Kenneth, and Bailey, William C.: Danger in the Air: Occupational Health (television broadcast), Part 3 of the Health Field Series. Kansas City, Missouri: Communications Division, American Lung Association, May 1983.

6. Bailey, William C., and Vath Richard R.: Lung Cancer (videorecording), 60 minutes. Health Sciences Consortium, 1985.

7. Bailey, William C.: Asthma (videorecording). Lifetime Television (Cable), 1986.

8. Bailey, William C.: Tuberculosis (videorecording). Lifetime Television (Cable), 1986.

9. Bailey, William C.: Use of Steroids in Asthma (audiotape). Southern Medical Association Dial Access Program, 1988.

10. Bailey, William C.: The Appropriate Use of Inhaler Therapy in Asthma (audiotape). Southern Medical Association Dial Access Program, 1989.

11. Bailey, William C.: The Use of Inhaled Steroid Therapy (audiotape). Southern Medical Association Dial Access Program, 1989.

12. Bailey, William C.: Emergency Room Treatment of Asthma (audiotape). Southern Medical Association Dial Access Program, 1989.

[Plaintiff's Exhibit #2]

THE HEALTH CONSEQUENCES OF SMOKING

CANCER AND CHRONIC LUNG DISEASE IN THE WORKPLACE

a report of the Surgeon General

U.S. Department of Health and Human Services
Public Health Service
Office on Smoking and Health
Rockville, Maryland 20857

• • • •

TABLE 5.—Comparison of smoking habit data obtained during life and after death

Smoking habit data obtained during life, 1971	Number	Smoking habit data obtained at death			
		Never smoked	Formerly smoked	Smoked	Smoked at some time
Never smoker	12	8	2	2	0
Ex-smoker	26	2	15	2	7
Smoker	76	1	12	33	30

SOURCE: Berry et al. (1985).

* * * *

[Plaintiff's Exhibit #3]

Reducing the Health Consequences of Smoking

25 YEARS OF PROGRESS

a report of the Surgeon General

1989

U.S. Department of Health and Human Services
Public Health Service
Centers for Disease Control
Center for Chronic Disease Prevention
and Health Promotion
Office on Smoking and Health
Rockville, Maryland 20857

* * * *

TABLE 11.—Estimated attributable risks for 10 selected causes of death from cigarette smoking, males and females, United States, 1985

Cause of death	Males (%)	Females (%)
CHD, age <65	45 (40-50)[a]	41 (34-48)
CHD, age ≥65	21 (17-26)	12 (9-15)
COPD	84 (78-88)	79 (73-83)
Cancer of lip, oral cavity, and pharynx	92 (79-97)	61 (45-76)
Cancer of larynx	81 (57-93)	87 (56-97)
Cancer of esophagus	78 (62-89)	75 (57-87)
Cancer of lung	90 (88-92)	79 (75-82)
Cancer of pancreas	29 (18-43)	34 (25-44)
Cancer of bladder	47 (31-63)	37 (18-61)
Cancer of kidney	48 (32-64)	12 (3-43)
Cerebrovascular disease, age <65	51 (36-65)	55 (45-65)
Cerebrovascular disease, age ≥65	24 (16-35)	6 (2-14)

NOTE: Computed from Tables 2, 6, and 7. For adult men under 65, the proportions of current and former cigarette smokers in 1985 were, respectively, 34.7 and 25.8 percent. For men 65 or older, the prevalances of current and former cigarette smoking were, respectively, 19.4 and 51.1 percent. For adult women under 65, the corresponding proportions were 30.1 and 16.5 percent; for adult women 65 or older, 12.6 and 19.6 percent.

[a] Numbers in parenthesis are 95-percent confidence intervals

* * * *

TABLE 18.—Estimated risks of various activities

Activity or cause	Annual fatalities per 1 million exposed persons
Active smoking	7,000[a]
Alcohol	541
Accident	275
Disease	266
Motor vehicles	187
Alcohol-involved	95
Non-alcohol-involved	92
Work	113
Swimming	22
Passive smoking[b]	19
All other air pollutants[b]	6
Football	6
Electrocution	2
Lightning	0.5
DES in cattlefeed	0.3
Bee sting	0.2
Basketball	0.02

NOTE: Activities are not mutually exclusive; there are overlaps between categories. Differences in fatalities do not imply proportionate differences in years of life lost.

[a] Number of deaths per million smokers who began smoking before 1965.

[b] Cancer deaths only.

SOURCE: Active smoking, CPS-II; NHISs 1965, 1985; U.S. Bureau of the Census (1974, 1986). Other activities causes, U.S. resident (1987).

* * * *

[Plaintiff's Exhibit #4]

THE HEALTH CONSEQUENCES OF SMOKING FOR WOMEN

a report of the Surgeon General

Prepublication copy: This copy is issued preparatory to printing as a formal document and is subject to editorial change. It omits an index which will appear in the final issuance.

U.S. Department of Health, Education and Welfare
Public Health Service.

* * * *

TABLE 8.—Histologic Types of Pulmonary Cancers in Smokers and Non Smokers

		Male		Female	
Type	Total	Smokers	Non-Smokers	Smokers	Non-Smokers
Epidermoid	992	892	7	80	13
Small Cell	640	533	4	100	3
Adenocarcinoma	760	492	39	128	101
Large Cell	466	389	16	46	15
Bronchioloalveolar	68	35	4	13	16
TOTAL	2,926	2,341	70	367	148

SOURCE: Resenow and Carr (39).

* * * *

[Plaintiff's Exhibit #5]

OBS	Name	Hosp. No.	Age	Histlogy	Pri. Site	Sur-gery	Sur-gery
1		89-48-85	34	8430	1623	1	5
2		00801382	37	8140	1629	0	2
3		00818283	38	8481	1623	1	8
4		00740064	36	8140	1623	1	3
5		00861339	35	8140	1623	0	1
6		00866553	36	8070	1622	1	8
7		00864742	39	8140	1623	1	8
8		00883227	38	8070	1623	1	5
9		00902963	29	8070	1629	0	0
10		00882055	38	8070	1623	0	0
11		00921916	38	8070	1623	0	1
12		00940420	29	8070	1623	0	0
13		00958265	28	8250	1623	0	1
14		00596419	38	8140	1624	1	5
15		01001846	38	8070	1623	1	8
16		00549551	32	8310	1623	1	5
17		00549551	32	8310	1623	1	3
18		01039428	38	8012	1623	1	5
19		01014568	37	8070	1625	0	0
20		00698307	37	8041	1622	0	0
21		01061452	32	8010	1629	0	1
22		01092377	37	8430	1629	1	7
23		01092439	33	8070	1623	1	5
24		01142091	33	8240	1624	1	5
25		01154179	37	8070	1623	1	5
26		01128174	38	8010	1625	0	0
27		01161962	31	8140	1625	1	3

Lung/Bronchus

0 None
1 Biopsy Only
2 Exploratory/Palliative
3 Partial Lobe Resection
4 Segmental Lobe Resection
5 Lobectomy
6 Pneumonectomy, Nos
7 Radical Pneumonectomy
8 Surgery, Nos
9 Unknown

Surgery variable

0 = no surgery
1 = surgery at UAB

UNITED STATES DISTRICT COURT
NORTHERN DISTRICT OF GEORGIA
ATLANTA DIVISION

(Title Omitted in Printing)

EXAMINATION BEFORE TRIAL of the Defendant, GENERAL ELECTRIC COMPANY, taken by and through its representative, THOMAS O. ROUSE, held at the Desmond Americana, Albany-Shaker Road, Albany, New York on July 27, 1993, commencing at 9:10 a.m.; before Paula M. Marcucci, a Shorthand Reporter and Notary Public in and for the State of New York.

* * * *

[3] THOMAS O. ROUSE

having been first duly sworn by the Notary Public, [4] was examined and testified as follows:

* * * *

Q What is your professional address?
A General Electric, 100 Woodlawn Avenue, Pittsfield, Massachusetts.
Q You are manager of dielectric projects?
A Yes.

* * * *

[7] Q If you began work for GE in 1960, would it be fair to say that this has been your only full-time work as an adult?
A Since I left graduate school, yes.
Q And you went into graduate school fairly immediately after college?
[8] A Yes.

Q And you went into college fairly immediately after high school, or did you go in the service—

A No.

Q —directly after high school?

So that, has there been any other outside income after your PhD program was finished?

A Not for me personally.

* * * *

[9] Q When did you first have any knowledge, in your personal or professional capacity, of the use of PCBs as dielectric fluids?

A On my arrival in Pittsfield in April of 1973, when I left Corporate Research and Development and joined the transformer business.

* * * *

Q In the late '70s?

A The point I was getting at, about 99 percent of dielectric fluid used in transformers in the U.S. and the world is mineral oil.

* * * *

[13] Q When you first became involved, then, with the PCBs, they had already been terminated as an askarel or dielectric fluid by General Electric; is that right?

A No, General Electric stopped using PCBs in dielectric fluids around 1977—from '73 to '77. As I indicated, I was involved not as a primary activity, but I was involved with the search for substitutes in a peripheral way.

* * * *

[21] Q Do you know when General Electric first began to use PCB containing askarels?

A It's my understanding that was 1934.

* * * *

Q Why use PCBs containing askarel as opposed to non-askarel mineral oil dielectric fluid?

A It was found that PCBs do not burn easily and so in transformers they were used for that nonflammability application. That means, then, in transformers where a fire was a particular—could present a particular hazard if it began, askarel transformers were frequently used. Askarel filled transformers—and, as a matter of fact, the National Electric Codes list askarel transformers as one of the alternative ways of providing transformers in such areas, these are typically substations such that they are enclosed in buildings. They would be the basements of buildings such as hospitals, other areas of other buildings of [22] considerable concern. They had been used in manufacturing plants such as aluminum electrorefining plants, and they've been used in underground applications where the electrical network extended under the streets of a street; for example, Manhattan.

Q Is there any need for the use of an askarel transformer if the utility has no particular problems with space? Doesn't have any high-rise buildings, doesn't have any underground lines, doesn't have any electric furnace, that the transformer can't be put out at the back of the lot?

A There is no particular need that I'm aware of. Askarel transformers did develop an excellent service record, and I am told they've been used in areas where flammability was not at issue but the attempt to repair or replace would be difficult phyically so they've been used in applications where low maintenance, high reliability are of a particular interest.

Q With respect to the internal parts of transformers, let's take two transformers side-by-side, the kind that are mounted on telephone poles. Is there any difference between one that would be filled with [23] mineral oil and one that would be filled with askarel?

A I'm not aware of any pole mount transformers that were filled with askarel, but to the extent that there was—

Q Let me change my question, then. Let's say a small substation where there are, on the ground, the transformers of the size of a large refrigerator.

A To the best of my knowledge, there is no difference in the design of the cores and coil structure. There may be some difference in the external casing for the heat exchange surface, but the coil and core structure are often identical.

Q And does the use of an askarel allow the transformer, assuming identical core structures, to be rated higher than a mineral oil transformer with that same coil structure?

A I will give you my opinion about this as a chemist. I am not an engineer. . . . It is my impression that askarel transformers are usually less highly rated than oil filled because the askarel is a higher viscosity and poor transfer [24] medium. It is further my impression that askarel transformers less readily withstand very abrupt changes in voltage, and so they are used in areas where there is greater protection against lightning, more carefully rated switch gear, switch gear that would protect the transformer from unusual voltage surges. So, in general, I think it is my impression, as a non-engineer, that askarel transformers are used perhaps in more delicate roles than are oil filled transformers.

Q How do askarel transformers compare to dielectric vented or dielectric sealed transformers?

A Again, it is my impression that dielectric transformers offer very little overload capability. Transformers are frequently bought, I believe, on the basis of their name plate rating. That is, they are rated to be able to handle a certain kVA on a continuous basis. In many cases, a transformer purchased on that basis will, in the summertime, for example, have to handle a high air conditioning load and so it will be overloaded. It will be loaded a value higher than its name plate rating for some

relatively short periods of time. Oil filled transformers have a greater heat transfer capability, [25] and therefore they are better able to take those relatively short, heavy loads in excess of their design, their average design load. In doing that, they may be loaded to 150, 175 percent of their regular load. Dielectric transformers, as I understand it, do not, if very little, have any overloading capability. So, if you put a dielectric transformer into the same action as the oil filled transformers with those loads, you would either have to build one twice as big and let its unused capacity sit there the rest of the year, or sit there, or buy two.

Q When these mineral oil transformers are running at above-normal capacity, you mentioned the heat transfer capabilities. How hot do they get inside?

A Typical temperature inside, at the hot spot inside the coil, will be 100-110.

Q Degrees—

A Centigrade. Degrees centrigrade.

Q Get as hot as 180 degrees centigrade?

A Under extreme overload, and in some special applications where high power capability is required, yeah, as high as 180 might be reached.

* * * *

[28] Q What is the problem with oxidation?

A Problem with oxidation?

Q Yes, sir.

A Mineral oil is a mixture of hydrocarbon refined products, petroleum products, like motor oil, in that sense, and on the in-service, it tends to form, on oxidation, acid and sludge.

Q So, the lower the propensity to oxidate, the lower the the propensity to have acid and sludge?

A Yes.

Q Is PCB, in any of its congeners, an appropriate additive to mineral oil?

A Not that I know of.

Q Does it belong in mineral oil, dielectric fluids?

A It is not intended to be in any. General Electric uses, as a matter of fact, in the ASTM and in your local internal GE specifications there is a maximum of two parts per million PCB allowed. So, in a sense, the specification says there shouldn't be any measurable amount of PCB in the mineral oil.

* * * *

[29] Q Does PCB in mineral oil serve any beneficial purpose?

A PCB in mineral oil?

Q Yes, sir.

A Not that I'm aware of.

* * * *

[33] Q Do PCDDs have any place in mineral oil transformers?

A They should not. I am not aware of any finding that suggests PCDDs are formed by the chlorination of any of the hydrocarbons in transformer oil.

Q How about PCDFs?

A I am not aware of anything that should involve the chlorination of the hydrocarbons in mineral oil for PCDFs. On chemical grounds, I would think it even less likely.

Q With respect to PCDFs and PCDDs, do they serve any useful purpose when contained in a dielectric fluid?

A I'm not aware of any.

* * * *

[38] Q Do you know what level, what percentage of mineral oil transformers could be expected to have some level of PCB contamination?

A It has been estimated, in approximately 1988 or '89, that there was some 35 odd mineral oil containing—35,000,000 odd mineral oil containing transformers in service in the U.S. My recollection of that same study indicated that there was some 2,000,000 thought perhaps

to contain measurable quantities of PCBs and some 200,000 of those to contain in excess of 500 parts per million PCBs.

* * * *

[40] Q And do you know the results of that test?

A It is my impression that there were, on occasions, PCDF concentrations in nanograms per gram—

Q That's parts per trillion?

A Yeah. [No. Parts per billion.]

—found in some samples that were tested elsewhere under that program.

Q How about PCDDs in mineral oil?

A I do not recall whether there was a finding of PCDDs in those samples.

Q Can PCDFs be formed from PCBs—

A Yeah.

Q —and trichlorobenzene in askarel fluids?

A Your question was can PCDFs be formed?

Q Yes, sir.

A PCDFs have been found to be formed from the partial oxidation of PCBs. It is conceivable, but I would think extremely unlikely, for PCDFs to be formed from partial oxidation from chlorobenzenes.

Q Does the existence of mineral oil affect in any [41] fashion the likelihood of the formation of PCDFs from PCBs?

A Mineral oils are hydrocarbons that are relatively easily oxidated; which is gasoline or diesel, lower molecular weight materials. They, if vaporized with PCBs, will be partially oxidized and will be competing for the oxygen that is available; compete in a statistical sense. And so the amount of partial oxidated PCBs that go on will be decreased by the presence of the hydrocarbons surrounding them in a vapor base.

Q Which is a more successful molecule at picking up the oxygen needed to oxidize? The PCB molecule that needs one oxygen that would become a PCDF or the hydrocarbon molecules in the mineral oil that needs

multiple oxygens to become on fire or whatever is happening to it?

A Well, hydrocarbons are relatively much more easily oxidated than are PCBs; that's why they're commonly used as fuels in cars and furnaces, and so I would think that, as an example, transformer oil has a minimum flash point of 135-145, ASTM and in your GE specifications. That's the temperature in a particular test of geometry at which vapor from the [42] transformer oil is ignited. PCBs do not have a flash point, at least a higher molecular weight. PCBs do not have a flash point.

Q But what temperature do you understand the PCBs will begin to burn?

A I do not know.

Q These are PCBs chlorinated in the range that is used in askarels.

A I do not know any temperature at which they will begin to burn. The lowest temperature which I have seen in the literature reported in their formation—for the formation of PCDFs in the range of three, three-twenty centigrade.

MR. COCHRAN: 300 to 320?

THE WITNESS: 300 to 320 centigrade, which would be to 575 Fahrenheit.

Q With the maximum conversion current, around 600 centigrade?

A 550 to 600 degrees centigrade, I believe, is what the literature said.

* * * *

[47] THE WITNESS: This had to do with the estimated number of transformers in the country and the estimated population of transformers contained in a 50 to 500 parts per million. In August of 1989 there was a report written to EPRI entitled "PCBs Residue Transformer Carcasses." It was prepared by GE Pittsfield, and I authored it. As part of that work there was a survey conducted by the Research Planning Corporation a firm down in Washington, attempting to the estimate of numbers of transformers in service and the earlier numbers I gave you were in an attempt to reproduce the results of that study and were garbled a little bit. The studies conducted and published in 1989 estimated that [48] there were 20,000,000 mineral oil filled transformers in the country. There were 2,000,000 units containing 50 to 500 parts per million PCB estimated in service, and there were 220,000 units with greater than 500 parts per million. I think in my earlier comment I said something about 32,000,000 transformers, and I believe that's the number that's about right for today, 1993, for the total population, and since 1993 I would think that the number of units containing PCBs has decreased somewhat, as you would, pulled from service and not replaced or replaced with transformers, the units are replaced with transformers which do not have traces of the PCBs in them.

* * * *

[49] Q So, instead of the rough estimate we talked about earlier, 1 in 16, it's closer to 1 in 10?

A That's their estimate, yes.

Q And you think that sounds like a fair estimate?

A I'm sure they knew what they were doing in 1988. As I indicated a minute ago, I suspect that as older transformers are replaced in service, the number of transformers that contain traces of PCBs has gone down since replacement. New transformers do not have PCBs in them. I would think the numbers are somewhat lower today.

Q And the last date of potential contamination would have been 1977 when the industry stopped making PCB askarel transformers?

A Yes, and the regulation controlling the manufacturer [50] went into effect in '78. I'm sure there was action before that by the manufacturer.

* * * *

[60] Q How do PCBs get in mineral oil dielectric fluids?

A There are clearly several alternatives. One is that the oil used to fill transformers in manufacturing plants, mineral oil, is contaminated in some way by PCBs. A second is that the oil used to fill the oil—used for filling large transformers in the [61] field could contain PCBs, and many ultilities buy oil directly for use in transformers in their system and that oil might come from a refiner of new oil; it might come from small companies which took used oil from various sources, cleaned it up and resold it. And as a subcategory of that, many particularly large refineries have in their yards oil treating systems for clean up of oil which they will reuse, cleaned up oil that's generated in their system and will reuse it; be returned to use in transformers.

* * * *

[66] Q At what stage in that process do you believe the mineral oil transformers became contaminated with the PCBs?

A In those cases where mineral oil filled transformers were filled with oil containing PCBs, I would think that, in general, it would have come from contamination by the oil itself—of the oil itself by PCBs.

Q And where would the oil become contaminated in the process of its handling?

A The plant in Pittsfield is nearly 100 years old; was nearly 80 years old when it went out of business. The manufacture, the time period that Pyranol transformers, Pyranol, was used, involved some pipes; oil involved some pipes. Manufacturing capabilities moved around the plant through that time. It could have been contamination in the pipes when they hooked up that; had briefly been used to Pyranol, presently hooked up for use with oil. Could have been a case in which there was an inadvertent interconnection for some period of time. The storage areas for the two fluids were in the same vicinity near

the railroad or truck unloading site. It seems unlikely that there was ever a misunloading, but that can't be ruled out. [67] Transformers are filled and drained for various purposes. A transformer could be inadvertently filled with askarel and drained into an oil line; equally so, a transformer could be inadvertently drained with oil and drained into the Pyranol line. There is no intention to do this sort of thing because Pyranol is considered, I believe, more expensive than oil, so it's a relatively precious commodity. Oil defeats the nonflammability aspect, character, of Pyranol, and Pyranol defeats the economics of use of oil; oil means that you're adding a higher cost fluid to a lower cost fluid to no purpose. So, in all cases these kinds of actions of mixing were unintentional.

* * * *

[82] THE WITNESS: Okay. APC who were the contractors on this report, this section of the report, estimated 28,000,000 transformers, mineral oil filled transformers in 1982, 3,000,000 of them were in the 50 parts to 500 parts per million category, and 305,000 were in the greater than 500 parts per million category.

MR. COCHRAN: Thank you.

THE WITNESS: That's 1982.

* * * *

[84] Q Could it happen?

A Formation of PCDFs in oil filled transformers for PCBs?

Q Yes, sir.

A I would think it extraordinarily unlikely.

Q Could it happen if it's hit by lightning?

A I would think that quite unlikely.

Q Happen if it catches on fire?

A If there is combustion of the fluid or the fluid vapor, certainly.

Q Can it happen if it's heated with lamps, 3000 watt football field lamps?

* * * *

A The experiments that are reported in the literature, at least were reported rather exhaustively in the literature to the mid '80s, indicated that the formation of PCDF from PCBs was only—could be found only in temperatures in excess of 300 degrees—

[85] Q Centigrade?

A —centigrade; 300-320, in that. The boiling point for mineral oils is around 290 centigrade for the point—the transformer oil—boiling point for a transformer is around 290. There is a very substantial amount of organic vapor that, in any gas over liquid, that may be containing a small amount of PCDF which would have combusted readily and statistically would use up oxygen in place of its availability for PCDFs formation. In addition, as I indicated earlier, there is about as much paper in the coil as there is oil, and it seems to me very unlikely that paper would survive temperatures of 300 degrees without charring, blacking, so if it is a question of heating the liquid alone, I would think it unlikely and certainly if it's a question of heat coil system involving both solid and liquid in solution.

Q If the PCBs are in solution with the mineral oil, as the mineral oil boils off first, vaporizes first, would the remaining PCBs—would the remaining oil be rich in PCBs or leaner in PCBs?

A The boiling range for mineral oil runs from around 290 to around 450-500 centigrade. The boiling range [86] for PCBs is really not too far different from that, but the boiling range, as I recall it, for 1260 Aroclor is somewhat higher than that. Therefore, I would think there is some possibility of a distillation separation occurring. If I can reiterate something I said earlier, if the insulation system involving paper is involved in that, I think

you would have other processes going on other than simple distillation.

Q Any of those processes create PCDDs?

A The charring of paper is not going to create PCDDs or PCDFs. It will create an unusable coil.

Q Can PCDFs be created and—PCDDs, I'm sorry, be created in a PCB contaminated transformer, meaning for my question, less than 1000 parts per million in mineral oil as a result of any foreseeable lightning strikes, fire, excessive heat, electrical arcing?

A There is no evidence that PCDDs are created from PCBs under any conditions.

* * * *

UNITED STATES DISTRICT COURT
NORTHERN DISTRICT OF GEORGIA
ATLANTA DIVISION

(Title Omitted in Printing)

EXAMINATION BEFORE TRIAL of a the DEFENDANT, GENERAL ELECTRIC COMPANY, through an expert witness, DR. JOHN BROWN, conducted pursuant to the Federal Rules of Civil Procedure, before Lillian M. Cunniff, a Shorthand Reporter and Notary Public in and for the State of New York, held at the Desmond Americana, Albany-Shaker Road, Albany, New York, on Monday, July 26, 1993, commencing at approximately 12:30 p.m.

* * * *

[6] DR. JOHN F. BROWN, JR.

called by the plaintiff, having first been duly sworn by the Notary Public, was examined and testified as follows:

* * * *

[7] Q. What is your present occupation?

A. My present occupation is Manager of Health Research at the named organization, General Electric Corporate Research and Development.

Q. How long have you been with General Electric?

A. Since 1950.

* * * *

Q. Give us the benefit of your education which enables you to serve in your present capacity?

A. I received a Bachelor's degree, a B.S. in Science in chemistry from Brown University in 1947; a Ph.D., also in chemistry, from MIT in 1959; and many years later, 1968 to 1969, took a post doctoral fellowship in clinical

pathology at the Upstate Medical Center in Syracuse, New York.

[8] Q. Other than that stint in clinical pathology, have you had any formal training in medicine?

A. No.

Q. What was your doctoral thesis concerning?

A. The title was "Polyfunctional Catalysis," which was concerned with the development of synthetic chemical models for biological enzymatic processes.

Q. Is that in any way relevant to our discussion today concerning PCB's—the effect of PCB's on human health and the analysis of the existence of PCB's in human adipose tissue. And when I say PCB's, I have included known contaminants, such as PCDD's and PCDF's.

A. Only to the extent that my thesis was concerned with the rates and mechanisms of chemical reaction. And after many years of research on diverse subjects, I have been concerned with, among other things, the rates and mechanism of clearance of PCB's from the human body and the information they provide as to the mechanisms of a body's response to the PCB's.

Q. What other major papers—papers that you would consider major—have you been involved in preparing as an author? Do you maintain a list of those, by the way?

A. Yes. I have written about 60 technical papers.

Q. How many of those relate to PCB's?

[9] A. Most of those written in the past dozen years.

* * * *

[21] Q. What is a PCB?

A. PCB stands for polychlorinated biphenyl. It's a set of initials that did not come into use until the late 1960's, and was not so designated until earlier. It can refer either to the family as a whole, or one of the individual commercial products, or to one of the individual chemical components of one of those products.

Q. Let's talk about the chemical individual first, and then we can break it down into the other topics you mentioned. How many congeners of PCB do you recognize?

A. 209.

* * * *

[24] Q. Do you believe there is any association between PCDD and PCB; that is, a PCB in any fashion—sunlight, aging, fire—anything that can convert PCB's to PCDD's?

A. I do not believe so.

Q. What about PCDD's from other ingredients in commercial askarels; for example, Pyranol has—tricholorobenzyne is a major constituent, about 40 percent of that; correct?

A. Yes.

Q. Can PCDD be formed from the askarel mixture which includes tricholorbenzynes through fire, aging, sunlight, or any other action of which you are aware of?

A. Yes. I am aware that there have been scientific reports of the conversion of chlorobenzynes to trace quantities of PCDD's at high temperatures.

Q. Do you know what temperature is necessary to make that conversion occur?

A. I do not know what is reported in the literature on that conversion, no.

Q. Does that make scientific sense to you, not just that you have read it, but does it make scientific sense to you that, yes, indeed PCDD's can be caused to come into [25] existence from a commercial askarel mixture under certain circumstances?

A. Yes. But it would depend on what else the askarel was mixed with. If the askarel was mixed with transformer oil, this would become much less probable.

Q. Transformer oil, meaning standard mineral oil used in transformers?

A. Yes.

Q. Why is that?

A. Because the oxidation of a chlorobenzyne to dioxin involves the capture of reactive oxygen fragments in the flame formed at high temperature. And these would be much more easily captured by hydrocarbon molecules than chlorobenzyne molecules.

* * * *

[26] Q. [. . .] What do you consider the most commonly and widespread PCDD congener that you know?

A. The most widespread PCDD congener is octachloro dibenzo dioxin, which is a fairly common combustion product.

Q. Combustion of what?

A. Of any organic material containing any chlorine compound.

Q. Including PCB's?

A. Including salted wood.

Q. But I need you to answer the first question. It does include PCB's; is that correct?

A. Yes.

* * * *

[31] Q. But does that make scientific sense to you, that if metal turns red hot and PCB's are on it or near it to gather its heat at close to 600 degrees centigrade, that those PCB's will begin to convert to PCDF's?

A. Yes, in the presence of oxygen.

Q. Does mineral oil—the existence of mineral oil affect this conversion rate as you described it affects, in your opinion, the conversion rate of PCDD's?

A. In my opinion, the mineral oil would inhibit PCB oxidation to dibenzo furan under any conditions.

* * * *

[38] Q. Do you believe that PCB's have any toxicity to human beings as contained in askarel dielectric fluids?

A. We have spent the last 16 years looking for such effects in people with heavy occupational exposure without finding any evidence of deleterious health effects.

Q. Do you not consider chloracne a deleterious health effect?

A. We do not believe that chloracne has ever been seen in people who were exposed to unaltered electrical grade PCB's.

Q. What is an alteration that you think would be necessary to electrical grade PCB's to cause them to give a human being chloracne?

A. Prolonged exposure to high temperatures of 300 degrees C.

Q. How long?

A. Days.

* * * *

[39] Q. When you decide to conduct a scientific experiment, do you prepare a protocol or a plan for doing so?

A. Not normally.

Q. Do you develop a question that needs to be answered or a hypothesis that needs to be proven of disproven?

A. Yes. I think it is essential to the nature of scientific investigation that one attempt to distinguish between alternative hypotheses as to the causation of the phenomenon, if any, being observed.

Q. So, eighth grade science students would be familiar with the idea of formulating the hypothesis that water freezes at 32 degrees Fahrenheit; and then, they would be about trying to prove that hypothesis. Is that an example of that procedure?

A. Yes. But the difference between professional research and eighth grade science experiments is that the [40] identification of the key hypothesis is very frequently a major portion of the research investigation to be conducted.

Q. And very frequently that identification is affected by the person financing the investigation; is that correct?

A. I would not say the identification, because the difference between scientific and advocacy procedures is

that basically in a scientific investigation, one is attempting to gather information to eliminate alternatives rather than argue for a particular hypothesis.

Q. But isn't it true that General Electric has never paid you to prove that PCB's are toxic, but instead has paid you to prove that PCB's are not toxic?

A. I have not received any instructions as to how this research—what the findings should be. My involvement in this program came at my initiative, not General Electric's, because I felt it was important to find out. It is also true that the people in the Capacitor Department who initiated the study were the plant hygienists, who was the nurse, and the physician who had a part-time job, whose major job was as a general practice physician in the community, who were very much concerned as to possible health effects on the people for whom they were responsible.

[41] Q. People for whom they would be responsible under worker's compensation statutes if, in fact, there was a causal effect between any illnesses suffered by these people and their job place exposure to PCB's; is that correct?

A. The orders that we got from—and on one occasion when we talked to the corporate medical director —were to find out what the problem was all about. They said they were fully prepared to pay workmen's compensation claims to people who needed that, who deserved them, but they were not interested in paying such claims to the entire population of Upstate New York. That's as far as our instructions went.

Q. Did your instructions as a researcher for General Electric also include voluntarily going to the Government and arguing with them about their classifications of PCB's as potential carcinogens, or suspected carcinogens, or whatever classification the Government thought was appropriate compared to with what General Electric thought was appropriate?

A. I have never been told what General Electric thought was appropriate. I initiated these—I proposed these research programs in 1976 and '77, and spent two years convincing the Company that it was a good idea to carry on health effects research.

Q. But isn't it true that the Company did, in fact, [42] have a significant role, non-scientist, within the Company, such as vice-presidents had a significant role in the words you used when you communicated with the Government in writing?

A. No.

Q. That was never approved?

A. As far as I know, the words that I have used that have appeared under my name were those that I selected and put into my reports.

* * * *

[48] Q. In fact, you have never come to a therapeutic modality to treat someone with high levels of PCB's; is that correct?

A. Nor has anyone else.

Q. So, there is no known treatment?

A. There are some procedures that can be used to reduce PCB levels to a modest extent, but these would be therapeutically unacceptable to the medical community, because in the absence of any medically convincing evidene of health effects associated with existing PCB body burdens, it would be medically improper for a physician to prescribe such a treatment.

* * * *

[49] Q. In other words, if you have a drastic weight loss, it's your opinion that the effect is to reduce the half-time?

A. No. The treatment—the therapeutic—the pharmacological intervention that caused the weight loss in [50] this case results in PCB removal. This is not the normal case. There is a considerable body of research on experimental animals indicating that weight loss normally

results in an increase in the concentration of PCB's in lipid reservoirs in the body.

* * * *

[52] Q. Do you believe that warnings advising people not to inhale, ingest, or absorb PCB laden materials are appropriate?

* * * *

A. Such warnings are routinely given regarding almost all chemical materials.

Q. Are they necessary with PCB's?

* * * *

[53] A. Probably much less so than with most other materials. The acute toxicity is known to be very low.

Q. Do you hold that opinion even with the knowledge that PCB's often are contaminated with PCDF's?

* * * *

A. The question is incorrect. PCB's are not often contaminated with PCDF's. It's very rare for them to be contaminated with significant levels of PCDF's.

Q. Would you agree with me that it is foreseeable that in such instances PCDF's will exist at some level in PCB products, such as Aroclors?

* * * *

A. I think I already testified that PCDF's can be found in electrical grade PCB's, at levels of from zero to two parts per million in what we have seen, and that these levels are toxicologically insignificant.

* * * *

[54] Q. Do you think that death is a risk of exposure to PCB's?

[55] A. No.

Q. Do you think that death is a risk of exposure to PCDF's?

A. The available evidence in both cases comes from statistical studies that have been done on people exposed to either PCB's or PCDF's. To my knowledge, such studies which involve analyses of death records have not shown any elevated mortality in the case of people exposed to PCB's, with quite large populations involved in the studies. So, the statistical power is considerable.

In the case of PCDF's, the number of deaths that have been looked at by the Japanese in their review of the Yusho poisoning victims is considerably more limited. It is my understanding that thus far Dr. Kuratsune, who has been the head of the Japanese investigative team, has been reluctant to ascribe any increased mortality to the people in the Yusho poisoning group.

* * * *

[64] Q. So, all of your responses on that are going to be based on the Triangle Laboratory reports, because we are going to ask about that in a little while.

A. Okay. Then, I will say based on my observation of the Triangle Laboratory reports, he showed a level and distribution of PCB's that was in the background range; in fact, below the normal environmental background—excuse me, below average environmental background.

* * * *

[86] Q. Is there anybody in General Electric, to your knowledge, who has written more published articles about PCB's than you?

A. Well, I have never made a publication count, but I'm not aware of one.

Q. Do you think that you would be considered to be the person at General Electric who knows as much about PCB's as anybody there?

A. Yes, about PCB science and some of its technological aspects. But I had no knowledgeability about anything involving commercial practices.

Q. We are going to come down to the home stretch. You have mentioned technological aspects; so, let's go right [87] into this Triangle Laboratory report. Have you reviewed that relating to Mr. Joiner?

A. I have not reviewed the details of the analytic methodology, because my colleague, George Frame, agreed to take on that assignment. What I did do was to examine the level and distributions of the PCB and PCDF congeners found to determine as best I could whether they were within a normal or abnormal range.

Q. And what did you discover?

A. What I found was that the levels of PCB's and PCDF's were both within the lower part of the range normally involved in environmentally exposed populations, that there were no obvious abnormalities that might have resulted from accelerated clearance, that the homologue distribution in the PCB's were again very similar to what we and many others have seen in background exposed populations.

* * * *

[94] Q. But it would be your opinion at any rate that even if he showed instead of .4 or .3 something parts per million of PCB's, even if he showed 1 part per million, that would not necessarily be causing him any problem; is that right?

A. I would guess that the average individual sitting in this room has more than one part per billion—

Q. I said million.

[95] A. Million.

Q. Would you guess that the average person in this room has more than one part per million?

A. Probably close to that.

Q. That would be 100 parts per billion; is that right?

A. One part per million—no, in adipose tissue.

Q What would that be per billion?

A. That would correspond to about five parts per billion in serum.

Q. What would 100 parts per million of adipose tissue correspond to in parts per billion adipose tissue? I'm not trying to quiz you on your math skills; I'm asking so that I will be clear.

A. Wait a minute; 100 parts per million in adipose tissue?

Q. Yes.

A. That's a pretty high level.

Q. Because you and I were talking apples and oranges. Somehow, I got lost in here. You switched serums of adipose on me in the middle.

A. There is a 200-fold difference between the two; that's the way to think about it on the average.

Q We discussed at Dr. Schector's [*sic*] deposition that [96] there are folks who believe that .4 parts per million in adipose tissue is within the normal background range.

A. Yes.

Q. Do you agree with that?

A. Yes.

Q. At what level would you start to have concern about someone's physical health, keeping the scale at parts per million and keeping the tissue at adipose?

A. I don't know that, because we have not seen effects in people who probably had adipose tissue levels as high as a thousand parts per million.

* * * *

[97] Q. There is no PCDF testing available for Mr. Joiner is there in what you have reviewed?

A. I recall there were PCDF analyses reported on him.

Q. What was your opinion as to those levels?

A. The levels and distributions again were within the range that has been reported for the American population, and even the pattern of non-detects reported corresponds to what has been reported for a natural range of background population.

Q. How about PCDD levels in Mr. Joiner?

A. They were also low. I did not check on the distribution of non-detects.

* * * *

[101] Q. Are you familiar with the work of Arnold Schector [*sic*] on the subject of PCB's?

A. Arnold Schector [*sic*] has written a hundred papers on PCB's and dioxins, mostly dioxins. I have not read all of them.

Q. Do you consider him to be authoritative; his [102] papers?

A. Just the papers?

Q. The ones that you have read, did you find them to be consistent with scientific knowledge and authoritative with something published in your line?

A. Where he has reported data on observed environmental levels, the results are as good as the laboratories doing the work. I have known Arnold Schector [*sic*] for quite awhile. I'm rather skeptical of his interpretations, as are many other people, and I would not regard his interpretations of the analytical data he has as scientifically authoritative.

Q. Why not?

A. Because he is clearly a man who has written more than he has read. He is just cranking out paper without thinking about it.

Q. Do you think he has a bias?

A. Sure. He is trying to justify his position with respect to the Binghamton State Office Building fiasco.

* * * *

UNITED STATES DISTRICT COURT
FOR THE NORTHERN DISTRICT OF GEORGIA

(Title Omitted in Printing)

DEPOSITION OF STEPHEN B. HAMILTON, JR.

* * * *

[4] STEPHEN B. HAMILTON, JR., 154 Driftwood Lane, Trumbull, Connecticut, 06611, having been duly sworn, was deposed and said:

* * * *

[5] Q I've got a copy of your CV here. What did you do your thesis on in your Ph.D. program at Northwestern?

A I did research on aromatic heterocyclic compounds containing boron.

Q Does that subject have any relevance, other than [6] the analytic techniques you have learned, to this particular finding—does that have any relevance to your present knowledge of PCBs?

A No.

Q And you did some post-doctoral research in Germany. Was there any relevance in that study of the PCB issue?

A No.

Q Have you published any papers on the PCB issue or anything related to PCBs?

A I've been a co-author on several papers regarding PCBs.

* * * *

[33] Q What is it about your education and employment [34] background which you believe qualifies you to tell me about the health risks of PCBs?

A I think it's that I've established myself as a scientific scholar, having gotten a Ph.D. in chemistry, am familiar with research techniques, and have devoted the last about 12 years of my career to this particular issue.

* * * *

[39] Q You mentioned the term biodegradability a little while ago. Let me ask you another term: Bioaccumulation. Does that concern you from an environmental point of view with respect to PCBs?

* * * *

[40] A Bioaccumulation is not a toxic effect per se. It merely reflects the tendency of certain chemicals like PCBs to partition into certain media.

Q Can it have a toxic effect?

A Pardon me?

Q Can it have a toxic result?

A If the chemical is toxic to that species, it may have a toxic result.

* * * *

[60] Q Do you think PCBs can cause lung cancer?

A Do I? No.

Q Why not?

A Well, there have been a number of epidemiological studies of PCB-exposed populations, people with very high exposure to PCBs, and we have not seen excesses of lung cancer among those populations. In fact, we've generally seen deficits in lung cancer.

Q Are you aware of various studies which have shown [61] increases in lung cancer but which were found to be statistically insignificant, but nevertheless existed?

A With PCBs? Where the principal exposure was PCBs?

Q Yes, sir.

A I don't recall off the top of my head. I'm aware that if you look at this body of literature in aggregate, there are not excesses. In fact, in a study of our plant that awas done by an investigator from Harvard, sponsored by NIOSH, he attempted to see if there was correlation between several different cancers and PCB exposure, and the correlations were negative. In one case statistically significant that dealt with all cancers, negative but not statistically significant for lung cancer. By correlation, being negative means as PCB exposure went up, the incidence of lung cancer went down.

Q How about with PCDFs, do you consider it possible to get lung cancer from exposure to PCDFs?

A I don't have any information on that other than to say that the workers in our studies were exposed to the totality of what was in the PCB mixtures, which presumably contained PCDFs.

* * * *

[87] Q Do you believe that PCBs can promote any kind of cancer cells at any location in human bodies?

A I don't think there's any evidence to suggest that—

Q How—

A —in humans.

Q How about PCDFs?

A There's a much smaller data base, but, again, I don't see positive evidence.

* * * *

UNITED STATES DISTRICT COURT
NORTHERN DISTRICT OF GEORGIA
ATLANTA DIVISION

(Title Omitted in Printing)

EXAMINATION BEFORE TRIAL of a [] DEFENDANT, GENERAL ELECTRIC COMPANY, through an expert witness, DR. GEORGE FRAME, conducted pursuant to the Federal Rules of Civil Procedure, before Lillian M. Cunniff, a Shorthand Reporter and Notary Public in and for the State of New York, held at the Desmond Americana, Albany-Shaker Road, Albany, New York, on Monday, July 26, 1993, commencing at approximately 10 a.m.

* * * *

[4] DR. GEORGE FRAME,

called by the plaintiff, having first been duly sworn by the Notary Public, was examined and testified as follows:

* * * *

[6] Q. Am I correct in my understanding that your role here today is to testify concerning the adipose tissue testing done by Triangle Labs on adipose tissue of Robert Joiner?

A. Yes, sir.

Q. What is it about your education and training which you believe qualifies you to opine on this subject?

A. I have a Ph.D. in analytical chemistry from Rutgers University; I have worked subsequent to that for 25 years in analytical chemistry; I have specifically conducted PCB analyses by a variety of methods, including mass spectrometry both for other organizations as well as the General Electric Company.

Q. Let's talk about your education very briefly. Where did you attend college, sir?

A. My undergraduate college was Harvard College, Cambridge, Massachusetts.

Q. What was your major there?

A. Chemistry.

* * * *

[22] Q. With respect to this particular case, what do you understand the methodology used by Triangle Labs consisted of?

A. I understand it to have been a Triangle Labs specifically designed method using generally acceptable sample extraction, and preparation, and clean-up steps for adipose tissue, and subsequent analysis—depending on whether the analysis was for PCB's, or for the PCDF's, or PCDD compounds—by high resolution gas chromatography using as the detection method high resolution mass spectrometry. An additional feature of the analysis is the use of istotope labeled internal and surrogate standards.

* * * *

[23] Q. The test for PCB's on Robert Joiner's adipose tissue; what do you think that means? What was the result?

A. The material that was presented to me, and the summary tables of the analysis of polychloronated biphenyls, reports the data for a number of components. And I have looked at the way in which the data was obtained and the way in which it was presented, and I find nothing to criticize or to disbelieve about the quantities of PCB's that are reported.

I find that the design of the analysis specifically identifies a subset of individual PCB chemical [24] structures, which is, in fact, the set of isotope labeled internal standards which enable the analyst to determine and identify specific PCB's. The design of the analysis also detects other PCB congeners which may be present, and quantitates them against the isotope labeled internal standards, but does not specifically identify which congener they are.

The summary data of all the congeners which are detected and quantitated by the standards that Triangle Laboratories employs to determine that they have, in fact, detected and quantitated adequately a congener, when summed, produce a total PCB content detected by this method in this sample of 0.3 parts per million by weight of PCB's.

* * * *

[28] Q. Have you read any of his writings concerning PCB, PCDD, and PCDF levels in human adipose tissue?

A. No.

Q. Why not?

A. I have only heard his name very recently with respect to this case, and my involvement has been strictly to examine the material from Triangle Laboratories to determine if the analysis was performed properly. I have not become involved in the interpretation of the levels.

* * * *

Q. What do you think about Triangle Laboratory's levels of PCDD's found in Mr. Joiner's adipose tissue?

[29] A. I found nothing to criticize about the methods that were used. Clearly, looking at the summary table, most specific congeners that were analyzed were not detected above their limit of detection, but I found nothing in my study of the analytical levels to indicate that any error or improper analysis was done.

* * * *

IN THE UNITED STATES DISTRICT COURT
FOR THE NORTHERN DISTRICT OF GEORGIA
ATLANTA DIVISION

(Title Omitted in Printing)

DEFENDANTS' JOINT MEMORANDUM IN SUPPORT OF SUMMARY JUDGMENT

INTRODUCTION

Plaintiffs, Robert K. Joiner and Karen P. Joiner, contend that Mr. Joiner's alleged occupational exposure to low levels of polychlorinated biphenyls (PCB's)[1] while employed as an electrical transformer repairman caused his small cell lung cancer.[2]

At the heart of this toxic tort case is the threshold question of causation. In recognition of that fact, the parties agreed on a scheduling order that focused first on the issue of cauation and permitted the defendant to test, on a motion for summary judgment, whether the plaintiffs would be able to present (through admissible expert opinions) *prima facie* proof of causation sufficient to enable them to go forward with their claims. *See*, July 13, 1993 Order. Defendants, General Electric Company ("GE"),

[1] PCBs are man-made chemicals once used in a wide variety of industrial applications, including as an insulating fluid in electrical equipment.

[2] Small cell lung cancer is a certain type of lung cancer as distinguished from, for example, squamous cell lung cancer. According to one of the plaintiffs' experts, Dr. Larry Robertson, "there are a multiplicity of cancers arising from a number of different cell types, from a variety of different causes. That is general knowledge." (Robertson Depo., at 13).

Westinghouse Electric Corporation ("Westinghouse"), and Monsanto Company ("Monsanto"), submit this memorandum in support of their joint motion for summary judgment, on the following grounds:

SUMMARY OF ARGUMENT

In order to pursue a claim against these defendants, plaintiffs' hypothesize that Mr. Joiner's small cell lung cancer was caused by occupational exposure to PCBs. To support this hypothesis, the plaintiffs proffer the opinions of three "experts": Dr. Larry Robertson, Dr. Arnold Schecter and Dr. Daniel Tietelbaum. Although these experts admit that Mr. Joiner was a cigarette smoker and admit that cigarette smoke is a known cause of small cell lung cancer, the plaintiff's experts nonetheless attempt to link Mr. Joiner's small cell lung cancer to exposure to PCB's. Two of the plaintiff's experts state that Mr. Joiner's small cell lung cancer was "promoted" by PCBs after being "initiated" by cigarette smoke. A third expert states that cigarette smoke, PCBs, and various known carcinogens in Mr. Joiner's workplace combined in a "multifactorial mix" to cause Mr. Joiner's small cell lung cancer.

The plaintiffs' experts' opinions, however, are speculative, contradictory, hypothetical, unreliable, unhelpful, irrelevant and otherwise legally insufficient to provide plaintiff a prima facie showing on the issue of medical causation. The plaintiffs' experts' opinions fail for each of the following separate and independent reasons:

1. As admitted by plaintiffs' own experts, their opinions are not based on credible scientific evidence and are therefore "speculative."

2. The opinions are not supported by epidemiological studies. Epidemiological studies covering thousands of workers exposed to PCBs over several decades have not found any association between PCBs and lung cancer.

3. The opinions are based exclusively on isolated studies of laboratory animals. Laboratory animal studies, in and of themselves, are insufficient to constitute admissible evidence of causation in human beings because they are *not* reliable predictors of human health effects.

4. The opinions are based on erroneous facts and unfounded assumptions. The opinions did not "fit" the facts of the case, that is, they are not "sufficiently tied to the facts of the case". *Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 509 U.S. ——, 113 S.Ct. 2786, 2796, 125 L.Ed.2d 469, 481 (1993).

5. The opinions assume Mr. Joiner absorbed PCBs at a significant level. In fact, Mr. Joiner has a PCB body level *below* that of individuals in North America who have never been *occupationally exposed to PCBs.*

Because the opinions of plaintiffs' experts do not constitute admissible evidence upon which plaintiffs can rely to support their hypothesis, the plaintiffs have failed to establish a prima facie case of medical causation. By failing to establish a causal link between PCBs and small cell lung cancer, the plaintiffs cannot maintain this action. The defendants are entitled to judgment as a matter of law and their motion for summary judgment should be granted.

* * * *

IN THE UNITED STATES DISTRICT COURT
FOR THE NORTHERN DISTRICT OF GEORGIA
ATLANTA DIVISION

(Title Omitted in Printing)

DEFENDANTS' STATEMENT OF MATERIAL FACTS

In accordance with Local Rule 220-5(b)(1), the defendants, General Electric Company, Westinghouse Electric Corporation, and Monsanto Company, submit this separate and concise statement of the material facts to which they contend there is no genuine issue to be tried. This statement is submitted in support of the defendants' joint motion for summary judgment.

1.

Robert K. Joiner has worked at The City of Thomasville Water & Light Department ("Thomasville Water & Light") since 1973.

2.

As part of his employment at Thomasville Water & Light, Mr. Joiner worked with and around electrical transformers and voltage regulators.

3

The dielectric fluid contained in the electrical transformers and voltage regulators used by Thomasville Water & Light was mineral oil.

4.

After Thomasville Water & Light began testing its mineral oil-filled equipment for the presence of PCB contamination in 1983, it determined that less than twenty percent of its equipment was contaminated with PCBs.

5.

Of all of the equipment tested by Thomasville Water & Light, only about 2.5% of the transformers tested contained levels above 500 ppm, while only about 16.7% of the equipment contained levels above 50 ppm.

6.

The EPA considers any electrical transformer containing less than 50 ppm of PCBs to be a "Non-PCB Transformer" and does not regulate such equipment.

7.

One (1) transformer at Thomasville Water & Light (not manufactured by either GE or Westinghouse) contained 1,880 ppm PCBs, which was the highest reported level for any transformer or voltage regulator.

8.

In 1991, Robert K. Joiner was diagnosed with small cell lung cancer.

9.

Mr. Joiner smoked cigarettes, and both of his parents smoked cigarettes while he was growing up in their home; thus, according to the plaintiffs' experts, Mr. Joiner had consistent, substantial exposure to cigarette smoke for many years.

10.

Small cell cancer of the lung in humans is caused by tobacco smoke.

11.

According to the plaintiffs' own experts, there is no credible evidence as a scientific probability that PCBs cause or promote small cell lung cancer in humans.

12.

According to the plaintiffs' own experts, there are no epidemiological studies which conclude that PCBs cause or promote small cell lung cancer in humans, or more generally, any form of lung cancer in humans.

13.

According to a labtoratory test of Mr. Joiner's adipose (fat) tissue obtained by Mr. Joiner in 1991 approximately four months after the diagnosis of his small cell lung cancer, Mr. Joiner's body level of PCBs was .3 ppm.

14.

Mr. Joiner's body level of PCBs, as measured in the analytical laboratory test of Mr. Joiner's adipose tissue, shows that Mr. Joiner's body level of PCBs was slightly less than the average individual in North America who has not had any occupational exposure to PCBs.

15.

None of the plaintiff's expert witnesses can quantify either Mr. Joiner's exposure to PCBs or his dose of PCBs.

This 8th day of December, 1993.

/s/ Anthony L. Cochran
ANTHONY L. COCHRAN
Georgia Bar No. 172425
Attorney for Defendant
General Electric Company

CHILIVIS & GRINDLER
3127 Maple Drive, N.E.
Atlanta, Georgia 30305
(404) 233-4171

/s/ David H. Flint
DAVID H. FLINT
Georgia Bar No. 264600
Attorney for Defendant
Westinghouse Electric
Corporation

SCHREEDER, WHEELER & FLINT
1600 Candler Building
Atlanta, Georgia 30303-1845
(404) 681-3450

/s/ Joe C. Freeman
JOE C. FREEMAN
Georgia Bar No. 275700
Attorney for Defendant
Monsanto Company

FREEMAN & HAWKINS
4000 One Peachtree
303 Peachtree Street, N.E.
Atlanta, Georgia 30308-3243
(404) 522-0856

[Filed Jan. 31, 1994]

IN THE UNITED STATES DISTRICT COURT
FOR THE NORTHERN DISTRICT OF GEORGIA
ATLANTA DIVISION

(Title Omitted in Printing)

PLAINTIFFS' BRIEF IN OPPOSITION TO DEFENDANTS' MOTION FOR SUMMARY JUDGMENT

---

* * * *

TABLE OF CONTENTS

TABLE OF CONTENTS—Continued

TABLE OF CONTENTS—Continued

TABLE OF CONTENTS—Continued

* * * *

6. A PPM is More Than Enough to Kill.

Defendants' argument that Robert Joiner's exposure to PCBs was not significant, through their efforts to minimize the extent of their deadly chemicals, by analogizing the parts per million ("ppm") measurement with inches per mile, or pints per gallon, do not demonstrate the "vanishingly small nauture of a 'ppm' ". (Defendants' Brief at 4, fn 3) Instead, this demonstrates the incredible toxicity of these chemicals. A more relevant analogy is offered by Michael H. Brown, *The Toxic Cloud*, at pages 249-250. Mr. Brown writes that: "At about sea level there are approximately 10 billion trillion air molecules in a breath of air, or as many molecules as there are stars in the known universe. So one part per quadrillion, tiny as it seems, might translate into 10 million dioxin molecules in each breath."

The World Health Organization believes that "[n]o levels of PCBs . . . exposure that can provide an absolute assurance of safety can be identified on the basis of the available data." [86] No less an authority than the Administrator of the United States Environmental Protection Agency has concluded that "the concept of a threshold exposure level has no practical significance where carcinogens are concerned. This is due to in part to the irreversibility and long latency period of carcinogens." *Environmental Defense-Fund, Inc. v. Environmental Protection Agency*, 510 F.2d 1292, 1298 (D.C. Cir. 1975). Accordingly, there is no level of exposure which is not significant.[86]

[86] IPCS at 22.

* * * *

(2). The Testimony is Admissible Under Rule 104.

Rule 104(a) requires this Court to resolve "[p]reliminary questions concerning the qualification of a person to be a witness, . . . [and] the admissibility of evidence . . . ." Fed. R. Evid. Rule 104(a). Plaintiffs' experts are qualified and their opinions are admissible.

(a). Plaintiffs' Experts are Qualified.

The experts relied upon by Plaintiffs have impeccable credentials and are qualified to testify. Defendants have not seriously challenged their qualifications to testify.[84]

* * * *

[86] Put another way, no reasonable parent would ever intentionally allow their child to be exposed to any level of these chemicals if such exposure could be avoided.

* * * *

[84] Defendants have challenged Dr. Robertson's qualifications in some areas. This limitation is admitted by Dr. Robertson. Dr. Robertson's testimony, though relevant, is not necessary to defeat this motion. His testimony will assist the jury at trial.

IN THE UNITED STATES DISTRICT COURT
FOR THE NORTHERN DISTRICT OF GEORGIA
ATLANTA DIVISION

(Title Omitted in Printing)

AFFIDAVIT OF DR. ARNOLD SCHECTER, MD, MPH

Personally appeared before me, the undersigned officer duly authorized to administer oaths, Dr. Arnold Schecter, who after being duly sworn, deposes as follows:

INTRODUCTION

1.

I am Dr. Arnold Schecter, MD, MPH., a competent adult, laboring under no disabilities.

2.

I make this Affidavit for any lawful use or purpose. I have personal knowledge of the facts set forth in this Affidavit.

3.

I reside at 88 Aldrich Avenue, Binghamton, New York, 13903. My professional address is Clinical Campus, College of Medicine, SUNY—Health Science Center Syracuse, 601 Gates Road, Vestal, New York, 13850.

EXPERTISE

4.

My current curriculum vita is attached to this affidavit as Exhibit "A" and it accurately reflects my training and experience in the fields of medicine, public health, and the effects of toxic chemicals, specifically polychlorinated biphenyls ("PCB"), polychlorinated dibenzofurans ("di-

benzofurans" also sometimes referred to as "PCDF"), and polychlorinated dibenzo-p-dioxins ("dioxins" also sometimes referred to as "PCDD") on human beings. My research and training includes study of these chemicals in human beings.

5.

I am a tenured full professor of preventive medicine at a major New York State medical school, and have been for over fourteen years. Before that, I was on the faculty at the New Jersey Medical School, SUNY Downstate Medical School and Harvard Medical School. I am board certified in preventive medicine and have been since 1976. I am a Fellow of the American College of Preventive Medicine, the American College of Physicians, and also the American Association of Occupational and Environmental Medicine.

6.

I am licensed to practice medicine in Kentucky, New Jersey and New York. I am on the staff of all three hospitals in my county. I see patients, but primarily do research now, on patients, the environment, and laboratory animals. My research is focused on the toxic effects of dibenzofurans, dioxins, and PCBs. I work full time in this field, and am one of the few American physicians to do so.

7.

I am a member of several national or international epidemiology scientific societies and am also a member of several toxicology societies. I have published over 100 papers relating to dioxins, dibenzofurans and/or PCBs since 1981. My writings concerning dioxins, dibenzofurans and PCBs have been subjected to peer review and have been cited by other researchers and the World Health Organization. I have published collaborative scientific articles in peer reviewed journals with colleagues from other main line or traditional universities and institutes from the disciplines of chemistry, toxicology, epidemiology and oc-

cupational medicine. These scientists work at Columbia University School of Public Health, SUNY Buffalo Medical Center, the University of Rochester Medical Center, the University of Amsterdam in Holland, and University of Helsinki in Finland, the University of Umea in Sweden, Health and Welfare Canada, Mt. Sinai Medical Center in New York City, Midwest Research Institute, ERGO Laboratories in Hamburg, Germany, the food analysis laboratory in Norwich, England of the British government, the food analysis laboratory of North Rhine Westphalia in Germany, the University of Nebraska, the Department of Public Health of Michigan and the Agent Orange Commission of Massachusetts, the Academy of Sciences of China, and the Academy of Sciences of the former Soviet Union, the Hanoi and Ho Chi Minh City (Saigon) medical schools, and Daiichi College of Pharmaceutical Sciences in Japan, concerning the Japanese PCB and dibenzofuran rice oil incident.

8.

I have served as peer reviewer, consultant, and invited attendee at meetings of the World Health Organization which concerned dioxins, dibenzofurans and PCBs on a number of occasions during the past 10 years. At these meetings, I have supplied my own original scientific data on human tissue levels of these chemicals for World Health Organization publications.

9.

I have recently served as peer review for the Agency for Toxic Diseases and Substance Registry of the Centers for Disease Control and Prevention of the US Public Health Service for their Toxicological Profile documents on (a) PCBs, (b) dibenzofurans, and (c) dioxins.

10.

I served as participating scientist to the U.S. Environmental Protection Agency ("EPA") for their "Human Health Assessment" document for the dibenzofurans and

th[ei]r "Bioavailability of Dioxins" document and prepared written and gave verbal reports to the EPA for its current review of human health assessment of dioxin. I presented my research findings and recommendations to the Agent Orange and dioxin review committee of the Institute of Medicine of the National Academy of Sciences, part of which was incorporated into their final report published in 1993.

11.

I have presented numerous papers at most of the recent International Dioxin Symposia during the past 10 years and also presented at the American Public Health Association national meetings, and other meetings including the American Association for the Advancement of Science. The subjects were dioxins, dibenzofurans and/or PCBs.

12.

I have performed original dioxin, dibenzofuran and PCB research in Vietnam for over 10 years, the former Soviet Union since 1988, and Japan for a number of years. I was the principal investigator for an Agent Orange and dioxin project for the Agent Orange Commission working with the Michigan Department of Public Health and also for the Massachusetts Agent Orange Commission[.] These were subsequently published in peer reviewed scientific journals.

13.

I serve, or have served, as reviewer for a number of scientific journals in the past few years. At present, I am co-chairman of the research committee of the Association of Teachers of Preventive Medicine, an association of medical school faculty members in preventive medicine. I also serve as a medical consultant for the American Legion and advise them on dioxin and Agent Orange issues.

14.

I currently serve, and have served for over 10 years, on my medical school's research committee. I served for two years as a National Institutes of Health Research Fellow in Anatomy at Harvard Medical School, and one year as a National Institutes of Health Clinical and research fellow and Instructor in Medicine at Massachusetts General Hospital of Harvard Medical School.

15.

I served two years in the US Army Medical Corps during the Vietnam war, and was honorably discharged as a major. I serve as scientific and medical consultant to the American Legion on matters of medicine, Agent Orange and dioxins.

16.

I served for between two and three years as director of the Broome County (New York) Health Department. During this period, the State of New York Building in Binghamton, New York, had a fire involving a transformer in its basement. The transformer contained PCB dielectric fluid. I saw first hand the public health consequences of such a fire and also learned first hand about the consequences of burning or heating of PCBs which include the creation of dibenzofurans and dioxins. As noted above, I have done extensive research into this area since that time. As an aside, the fire caused very little property damage but the contamination caused by the PCBs, PCDFs and PCDDs has kept the building unsafe and vacant for more than 10 years and over forty million dollars has been spent on cleanup efforts.

OPINION

17.

I have been asked to render an opinion as to the causal relationship between Robert Joiner's exposure to PCB

contaminated mineral oil dielectric fluids and the lung cancer from which he suffers. It is my opinion, to a reasonable degree of medical and scientific certainty, that Robert Joiner's exposure to PCB contaminated mineral oil dielectric fluid is the cause of his lung cancer. I believe that Robert Joiner's exposure to PCB contaminated mineral oil dielectric fluid served as a promotional effect on his lung cancer cells, probably initiated by cigarette exposure years before and caused him to contract lung cancer at the very young age of 37. But for Robert Joiner's exposure to PCB contaminated mineral oil dielectric fluid I believe he would not now be suffering from lung cancer.

METHODOLOGY

18.

In reaching the opinions set forth above I have followed the methodology usually and generally followed by physicians and scientists. This methodology included at least the following steps:

A. I interviewed Robert Joiner and discussed with him his general health, his medical history, his family medical history including but not limited to other victims of cancer, his exposure to other potential carcinogens including but not limited to tobacco, his work history, and his specific tasks at work with special interest in his work involving transformer repair.

B. I reviewed Robert Joiner's deposition testimony and a sworn statement he had provided to me.

C. I reviewed Robert Joiner's medical records before and after he was diagnosed with lung cancer. Included in this review was a review of the adipose tissue testing performed by Triangle Laboratories and a discussion with the chemist who performed the tests of the methodology he followed.

D. I reviewed a videotape which illustrated the conditions in which Robert Joiner worked while repairing transformers.

E. I reviewed the results of testing, (only tests performed after 1986 were available), for PCB on transformers owned by Robert Joiner's employer which were representative of the transformers on which Robert Joiner worked and to which he was exposed.

F. I reviewed the (toxicological and biochemical) scientific literature (clinical and epidemiological and wildlife) on animals, humans which has been published in the areas of PCBs, dibenzofurans, and dioxins, a sample of which is discussed below. Additionally, I have reviewed the scientific literature which was presented or discussed at scientific meetings I have attended in the past ten years, or that is with the documents in paragraphs 9 and 10 above.

G. I reviewed the deposition testimony of Plaintiffs' experts—Dr. Daniel Teitelbaum and Dr. Larry W. Robertson, Ph.D. as well as the deposition testimony of Defendants' experts—Dr. Phillip Cole, Dr. William Bailey, and Dr. William Waddell, Ph.D. I have aso reviewed the affidavit of Dr. Arthur Frank, another of Plaintiffs' experts.

H. I relied on my own personal training and experience in that I have devoted the last ten years of my life to the study of dioxins, dibenzofurans and PCBs and am uniquely qualified to opine as to health effects, including the promotional effects on initiated cancer cells, of these chemicals.

I. I eliminated other causes, to a reasonable degree of medical certainty.

J. I know that PCBs, dioxins and dibenzofurans and their metabolites tend to accumulate in lung tissue, as demonstrated in the scientific literature in a number of countries. The lungs are the organ most affected in Robert Joiner.

This methodology allowed me to understand the carcinogenic potential of the chemicals to which Robert Joiner

was exposed, and allowed me to understand and discover the mechanism which caused Robert Joiner's lung cancer.

EXPLANATION

19.

PCBs are synthetic or man-made chemicals, which are very persistent in the environment and in humans. They are toxic to humans and animals. PCBs did not exist until they were manufactured, primarily since 1929. PCBs are very similar chemicals to dioxins and dibenzofurans, which are also very toxic synthetic chemicals. They are frequently found together. They are all very persistent and highly toxic, although of the several hundred congeners of these chlorinated chemicals, there are some which are much more toxic than others; similarly, some are not as toxic or as persisent as others. The higher chlorinated PCBs typically used in dielectric fluids (Aroclors) were typically of the more toxic variety. We learned from Christopher Rappe and H.R. Buser, European chemists, that when PCBs burn in the presence of oxygen they form much of the more toxic dibenzofurans and some dioxins. When the chlorinated benzenes sometimes found in PCB transformer fluid burn, they yield higher amounts of dioxins and lesser amount of dibenzofurans. Dibenzofurans are usually found in PCB transformer fluids, as reported in numerous reports and at many scientific meetings. In the Binghamton, New York transformer fire, we found PCBs, dibenzofurans and dioxins in the soot and in the air after the fire. This mixture was and is regarded as highy toxic and dangerous to human health, by the New York State Department of Health, the U.S. EPA and the Broome County (NY) Health Department.

20.

Dioxins, PCBs and dibenzofurans have been shown to be toxic at extremely low levels in humans, in wildlife and in animal experiments. They cause cancers, adverse re-

productive effects including malformations, immune deficiency or the inability to fight cancers and infections, liver damage, nervous system damage, endocrine system damage and other health effects. They may be complete carcinogens, as they act in animals; but certainly are also at least potent promoters of cancer in animals and humans. This has repeatedly been reviewed by Huff at NIH and Lucier at NIH. Recent work at NIH shows no threshold for various biochemical endpoints tested by Lucier and colleagues. The rice oil poisoning incidents in Japan in 1968 and Taiwan in 1979 show that small amounts of PCBs and dibenzofurans can cause very serious health effects, including malformations, cancer death and learning disabilities. These health effects have been shown by Masuda and Kuratsune and colleagues in Japan. Consistent with this is the work of Walter Rogan of NIH who studied PCBs in North Carolina and the rice oil incident of Taiwan with Drs. Shu, Guo, Yu and others. Recent publication of dosing experiments of pregnant rats by Peterson and colleagues showed even the lowest dose employed produced brain and endocrine system damage to the offspring.

21.

Robert Joiner, by occupational history, was exposed at work, for a long period of time on a regular basis to PCBs and other transformer fluid contaminants routinely, after fires and high heat incidents, and when there was heating during the repair process. These exposures were above and beyond the exposures suffered by the general population. The exposure of the general population is now known to be higher than previously thought, so the total amount he was exposed to was greater than that from work alone. He, like all other Americans, was also exposed to many other chemicals including initiators of cancer. Therefore, the initiating and promoting effects of the amounts of the PCBs, dioxins and dibenzofurans was certainly enough to have contributed to his lung cancer, to the best

of my medical and scientific opinion. He breathed the chemicals, got them on his hands, absorbed some through the skin and ate food contaminated with some of these chemicals. These exposure mechanisms exposed Robert Joiner to PCB levels well above those experienced by the general population.

22.

I reviewed the methodology followed by Triangle Laboratories in its adipose tissue tests for dioxins and dibenzofurans. There are several problems with the analysis performed by Triangle Laboratories. I have published work concerning adipose tissue testing on several occasions, and am familiar with results from proper testing techniques. I discussed the techniques followed by Triangle Laboratories with the chemist who conducted the tests. I believe mistakes were made. I do not believe that a reasonable scientists could use these results to compare Robert Joiner to other humans. The results show no detectable levels of PCDDs in Robert Joiner's blood for congeners which are known to exist at detectable levels in adults in North America. This casts considerable doubt on the methodology followed and further convinces me that no reasonable and experienced dioxin medical scientists would rely on them.

In addition to problems with methodology, there is no control group to which Robert Joiner can be compared. In adipose tissue tests, the subject's results are compared to the general population (the "control") to determine how the subject compares. No study has been documented that examines subjects who have both experienced weight loss associated with cancer, chemotherapy associated with cancer, and radiation therapy associated with cancer, as well as been subjected to excessive PCB, dioxin or dibenzofuran exposure. As a result, there is no control group to which Robert Joiner can be compared. Accordingly, even assuming the results were analytically accurate, as a scientist I cannot rely on them for accurate exposure assessment.

This point was made in the 1993 Institute of Medicine-National Academy of Sciences Agent Orange Report.

Adipose levels which are low do not mean that higher levels did not exist at earlier times. This is because PCBs are metabolized and excreted from the body over time. The scientific literature bears this out. Perhaps the best documented group of non-occupationally exposed victims of PCBs are the survivors of the 1968 Yusho rice oil PCB and furan poisoning. Twenty-five years after unquestionably high exposures, many of these victims had PCB levels which had returned to those of the general population—or nearly so.

If the test results are valid, they prove that Robert Joiner's body is contaminated with PCBs. There is no normal level of PCBs in humans. Human beings should have absolutely zero PCBs in their systems.

23.

It should be remembered that the National Institute on Safety and Health (NIOSH), the official U.S. government worker research institute, in its late 1970's PCB Criteria Document, stated that there is no safe amount of PCBs. Since these chemicals have been shown to have non-threshold curves in recent biochemical studies at the National Institutes of Health, it seems reasonable to conclude that any additional amount of these chemicals can lead to increased risk of serious health damage.

24.

We know that the lung is the favorite site for finding metabolites of PCBs and related chemicals as well as the chemicals themselves. This has been shown by Nagayama, Masuda, Brandt and others. With a concentration of these carcinogenic compounds in the lung, it is reasonable to believe that, with long time occupational exposure without protection or with adequate protection, these chemicals

were related to the lung cancer which is killing Robert Joiner. We also know that these chemicals when given to animals, without other initiators of cancer, will, in a dose dependent fashion, cause cancer in rodents, a common test method used to indicate what may happen in humans. These include scientific research by Kociba, Norback, Kimborough, Anderson and many others. They also promote induction of the enzyme AHH in cell cultures, another test which paralles human toxicity for these and similar chemicals.

25.

In addition, we know that Bertazzi has found an excess of lung cancer in PCB workers in a peer reviewed published epidemiology study, *Cancer Mortality of Capacitor Manufacturing Workers*, Bertazzi et. al., American Journal of Industrial Medicine 11:165-176 (1987). Kuratune and other Japanese workers also reported an increase in lung cancer in male PCB and dibenzofuran rice oil exposed persons in Japan. (Ikeda, et. al., *A Cohort Study on Mortality of Yusho Patients—A Preliminary Report*, Fukuoka Acta med., 78:297-300 and other studies support this). Further, although I have not known about the Monsanto documents until called recently to my attention, these documents prepared by Dr. E. Mahboubi of The Eppley Center, and Frederick R. Johannsen, Ph.D. of Monsanto, suggest that Monsanto's PCB exposed workers also were developing higher rates of lung cancer than would otherwise be expected. We also have the recent Institute of Medicine of the National Academy of Sciences Agent Orange report of 1993 noting that the literature supports human cancer and human cancer mortality from dioxin and Agent Orange, and does not refute or is not complete enough to come to scientifically conclusive evidence concerning many health effects of dioxin and related chemicals (*Veterans and Agent Orange; Health Effects of Herbicide Used in Vietnam*, National Academy Press, 1993

Washington, DC). I believe this work too supports my opinions.

26.

It is known that risk of lung cancer for former cigarette smokers decreases to general population levels with time after cessation of smoking. The fact that, despite not smoking for years, Robert Joiner still developed lung cancer, speaks strongly for the cancer promoting influence of PCBs, dibenzofuran[s] and dioxins in Robert Joiner's case. Also, in summary, in my professional opinion, the weight of scientific evidence regarding health effects of the very similar PCBs, dioxins, and dibenzofurans, including wildlife, in vitro and in vivo toxicology, and human clinical case reports, clinical studies and epidemiology studies, together point to these chemicals as being carcinogenic in humans, probably by being both promoters and initiators.

/s/ Arnold Schecter
DR. ARNOLD SCHECTER, MD, MPH

Sworn to and subscribed before me this 28th day of January, 1994.

/s/ Sally Kirtland
Notary Public
My commission expires: 9/30/94

Curriculum Vitae

Arnold J. Schecter, M.D., M.P.H.

Office:

Clinical Campus
College of Medicine
SUNY—Health Science
Center Syracuse
531 Gates Road
Vestal, NY 13850

Home:

88 Aldrich Avenue
Binghamton, NY 13903

Marital Status:

Married, three children

Fellow:

American College of
Preventive Medicine
American College of
Physicians
American College of
Occupational and
Environmental Medicine

Medical Licensure:

Kentucky (License #5276)
New York (License #115690)
New Jersey (License #82199)

Mailing Address:

PO Box 1000
Binghamton, NY 13902

Phone:

(607) 770-8521 or 8522
FAX (607) 770-8623

Social Security Number:

325-28-3836

Citizenship:

United States of America

Birth Date:

December 1, 1934

Diplomate:

American Board of Preventive
Medicine, 1976

Academic Position:

Professor
Dept. of Preventive Medicine
SUNY Health Science Center
Syracuse
Clinical Campus at
Binghamton

Hospital Medical Staff Positions:

Our Lady of Lourdes
Hospital
169 Riverside Drive
Binghamton, NY 13905

United Health Services
Hospitals:
PO Box 540
Johnson City, NY 13790

Education:

1974-1975 Columbia University School of Public Health, M.P.H., 1975

1958-1962 Howard University Medical School, Washington, D.C., M.D., 1962

1954-1957 The University of Chicago, Chicago, Illinois, B.A. (Liberal Arts) 1954, B.S. (Physiology-Neurophysiology) 1957

1951-1953 Shimer College of the University of Chicago, Mount Carroll, Illinois

Training:

1991 Mini-Residency in Occupational Medicine, University of Cincinnati College of Medicine, Part I

1982 Occupational Medicine Course, University of Illinois Medical School, Chicago, Illinois

1982 Occupational Medicine Course, Mt. Sinai Medical Center, New York, New York

1979 Occupational Medicine Course, University of Illinois Medical Center, Chicago, Illinois

1979 Environmental Medicine and Environmental Health Course, Postgraduate School, New York University Medical Center

1978 Occupational Medicine Course, Asbestos, Mt. Sinai Medical Center, New York, New York

1968 Course in Aviation Medicine, U.S. Army, Fort Rucker, Alabama

Professional Experience

1979-present Professor, with tenure, Department of Preventive Medicine, Clinical Campus at Binghamton, SUNY Health Science Center/Syracuse, College of Medicine, Binghamton, New York

1979-1981 Commissioner of Health, Broome County Health Department, Binghamton, New York also Medical Director, County Home Health Nursing Service, Acting Director of Willow Point Nursing Home, Binghamton, New York (part-time), Medical Director, Home Health Nursing Service

1975-1979 Clinical Associate Professor, Department of Preventive Medicine and Community Health, College of Medicine and Dentistry of New Jersey, New Jersey Medical School, Associate Director, Office of Primary Health Care Education, Office of the Dean, 1976-79

1972-1975 Assistant Professor, Dept. of Psychiatry, Director of Clinical Research in Drug Abuse; Coordinator and Faculty Member, Career Teacher Training Center in Drug and Alcohol Dependence Career Teacher Training Center and Division of Alcoholism and Drug Dependence, Drug and Alcohol Abuse Treatment Programs, State University of New York, Downstate Medical Center University of New York Health Science Center at Brooklyn and Kings County Addictive Disease Hospital, Brooklyn, New York

1971-1972 Director, Inpatient Rehabilitation Center, Drug and Alcohol Rehabilitation Program, Kentucky Region Eight Mental Health and Mental Retardation Board, Inc., Louisville, Kentucky

1969-1970 General Practitioner, West Point, Kentucky and Senior Aviation Medical Examiner (Federal Aviation Administration designee)

1967-1969 Captain, then Major, United States Army Medical Corps, Fort Knox, Kentucky

1966 Surgical Intern, Beth Israel Hospital, Boston, Massachusetts (January 1-December 31 Dr. Jacob Fine, Chairman)

1964-1965 Instructor, Department of Medicine, Renal Unit and National Institute of Health sponsored Clinical and Research Fellow; Harvard Medical School of the Massachusetts General Hospital, Boston, Massachusetts (Dr. Alexander Leaf, Director)

1962-1964 Postdoctoral Research Fellow, Harvard Medical School, Boston Massachusetts, 1962-64, funded by National Institute of Health, Department of Anatomy (Renal Ultrastructure, Dr. Don Wayne Fawcett, chairman)

Recent National and International Scientific Panels and Awards

Advisor to the Government Accountability Project, 1993

American College of Preventive Medicine's representative to the Physician Consortium on Substance Abuse Education, U.S. Public Health Service, 1992-present

Advisor to the Natural Resources Council of Maine, 1992

Technical Review Panel of the Great Lakes Protection Fund, 1990-present

Advisor to the National Veterans Legal Services Project, 1990-present

Invited member of the "U.S. Baikal (Russia) Research Association," 1991-present

Advisor to The American Legion's Science Panel, 1991-present

National External Science Advisory Panel of the Michigan Agent Orange Commission, 1989-present

Advisor to the National Veterans Legal Services Project, 1989-91

Advisor to the Environmental Defense Fund, 1984-89

Awarded the "Pacesetter Award" from the Commonwealth of Massachusetts for dioxin research, 1987

Temporary consultant to the World Health Organization of the United Nations on Chlorinated Dioxins and Related Chemicals in Human Breast Milk. Meetings at: Oslo, Norway, October 1986; Copenhagen, Denmark, November 1986 and February 1988; Toronto, Canada, September 1989; Rovaniemi, Finland, June 1990; Bayreuth, Germany, September 1990

Peer Review Committee (Expert Panel), USEPA *Biological Health Assessment Document, Polichlorinated Dibenzofurans,* 1986

Peer Review, USEPA, *Chlorinated Dioxins and Furans in the General U.S. Population: NHATS FY87 Results,* 1991

Contributor to "Bioavailability of Dioxins," *U.S. Environmental Protection Agency 1984 Workshop Proceedings,* Government Printing Office, 1985

Planning Committee, International Symposium on Dioxins and Related Chemicals, 1984, 1985

Recent National and International Scientific Presentations

Institute of Medicine, National Academy of Sciences, Scientific Workshop on Exposure Assessment, 1992

National Institute of Environmental Health Sciences and Health and Welfare Canada, Breast Cancer Prevention: Opportunities for Research, 1992

New York Chapter, Vietnam Veterans of America, 1992

Michigan Agent Orange Commission Meetings, 1991-1993

American Chemical Society National Meetings, 1983-1991

International Symposium on Dioxins and Related Chemicals, 1984-1992

American Public Health Association Annual Meetings, 1985-1991

American College of Preventive Medicine, Annual "Prevention" meetings, 1985-1991

American Association for the Advancement of Science, 1987

Prevention '86, Coordinator: Chemicals in the Environment: What Can Be Done?, Dioxins and Related Chemicals as a Model, 1986

Banbury Conference, invited speaker, Cold Spring Harbor, NY, 1983 and 1990

Gordon Conference on toxicology of dioxins, 1983 and 1985

Wisconsin Vietnam Veterans of America National Meeting, 1989

Academic/Professional Memberships

Association of Teachers of Preventive Medicine

Association of Teachers of Preventive Medicine Research Committee

American College of Epidemiology

American College of Occupational and Environmental Medicine (Fellow)

American College of Preventive Medicine (Fellow)

American College of Physicians (Fellow)

American College of Preventive Medicine Physician Consortium on Substance Abuse Education

Association of American Medical Colleges (1976-1990)

American Association for the Advancement of Science

American Public Health Association

American Society for Cell Biology (1962-1990)

Broome County Medical Society

New York Academy of Sciences

New York State Medical Society

New York State Occupational Medicine Association

Massachusetts General Hospital Housestaff Alumni Association

Microscopy Society of America

Society for Epidemiologic Research

Society for Occupational and Environmental Health

University Committees

Executive Committee, Clinical Campus, 1992-present

Grades Committee, 1992-present

Research Committee, 1979-present

Student Appraisal and Promotions Committee, 1979-1980

Search Committee, Department of Preventive Medicine, 1980-1983

Educational Policy Committee, 1979-1981, 1992-1993

Coordinators' Committee, 1979-1983

Library Committee, 1982-1987

Television Health Features Advisory Council, SUNY Binghamton, 1980-1981

Community Service

Broome County Nursing Home Advisory Board, *ad hoc*, 1979-1984

Broome County Mental Health Advisory Board, 1979-1984

Broome County Mental Health Advisory Board Subcommittee and Drug Abuse and Alcohol Abuse, 1983-1984

Broome County Health Department: Home Health Advisory Committee *ad hoc*

Broome County Health Department: Medical Advisory Committee *ad hoc*

Broome County Health Department: Advisory Board of Health *ad hoc*

Broome County Medical Society: Board of Directors, 1979-1984

Broome County Medical Society: Public Health Committee, 1979-1982

Broome County Water Resources Commission, *ad hoc* appointee

New York-Pennsylvania Health System Agency, Community Board

New York-Pennsylvania Health System Agency Planning Committee

Our Lady of Lourdes Hospital Hospice Advisory Board

Our Lady of Lourdes Hospital Hospice Finance Committee

Central New York Affiliate of the New York State Public Health Association

Planned Parenthood of Broome and Chenango Counties, Inc., Medical Advisory Committee

Broome County Chamber of Commerce

Broome County Chamber of Commerce Professional Committee

State Univerity of New York, Binghamton, Speakers' Bureau

Twin Tiers Home Health Nursing Service Board of Directors

BIBLIOGRAPHY

Editorial Activities

Cofounder and Associate Editor, *The American Journal of Drug and Alcohol Abuse*. New York, New York: Marcel Dekker, Inc., 1973-1978

Editorial Board, *The American Journal of Drug and Alcohol Abuse*. New York, New York: Marcel Dekker, Inc., 1978-present

Editorial Advisory Board, *Substance and Alcohol Actions/ Misuse*. Elmsford, New York: Pergamon Press, Inc., 1979-present

Reviewer, *Journal of Occupational Medicine*

Reviewer, *Journal of the American Public Health Association*

Reviewer, *Environmental Science & Technology*

Reviewer, *Academic Medicine*

Books

Schecter, A. (ed.). *Rehabilitation Aspects of Drug Dependence*. Cleveland, Ohio: CRC Press, 1977.

Schecter, A. (ed.). *Treatment Aspects of Drug Dependence*. West Palm Beach, Florida: CRC Press, 1978.

Schecter, A., Alksne, H. and Kaufman, E. (eds.). *Drug Abuse: Modern Trends, Issues and Perspectives*. New York, New York: Marcel Dekker, Inc., 1978.

Schecter, A., Alksne, H., and Kaufman, E. (eds.). *Critical Concerns in the Field of Drug Abuse*. New York, New York: Marcel Dekker, Inc., 1978.

Schecter, A. (ed.). *Biomedical Issues in Drug Abuse*. Volume I, Proceedings 1978 National Drug Abuse Conference. New York, New York: Plenum Publishing Co., 1981.

Schecter, A. (ed.). *Sociocultural Issues in Drug Abuse*. Volume II, Proceedings 1978 National Drug Abuse Conference. New York, New York: Plenum Publishing Co., 1981.

Schecter, A. (ed.) *Dioxins and Health*, Plenum Press, in preparation for 1993 publication.

Articles

1. Schecter, A.J. and D.W. Fawcett. (Abstract) "Intracellular microtubules in mammalian podocytes," *Anat. Record*, 148:2, 332, 1964.
2. Schecter, A. J. (Abstract) "Fine structure studies of the urinary bladder of bufo marinus," *Anat. Record*, 151:3, 412, 1965.

3. Schantz, A. and A. J. Schecter. (Abstract) "Iron hematoxylin and safranin O staining of epon embedded sections," *Anat. Record*, 151:3, 454, 1965.

4. Schantz, A. and A. J. Schecter. (Abstract) "The uptake of electron opaque markers in epithelial and mesothelial cells of the urinary bladder of bufo marinus," *J. Cell Biol.*, 27:94A, 1965.

5. Schantz, A. and A. J. Schecter. "Iron hematoxylin and safranin O as a polychrome stain for epon sections," *Stain Tech.*, 40:27, 1965.

6. Schecter, A. J. and M. J. Schecter. (Abstract) "A possible variation in unit membrane substructure," *J. Applied Physics*, 37:329, 1966.

7. Schecter, A. J. (Abstract) "The occurrence and distribution of intracellular microtubules in the mammalian kidney," In: *Proceedings, Third International Congress of Nephrology, 1966.*

8. Schecter, A. J. "Variations in unit membrane structure," *6th International Congress for Electron Microscopy Proceedings, II*, Tokyo, Japan: Maruzen Publishing Co., 397-398, 1966.

9. Schecter, A. J. "Variations in unit membrane substructure," *The Norelco Reporter*, XIII, 1966.

10. Schecter, A. J. "A different approach to a detoxification center," *Proceedings, Fourth National Conference on Methadone Treatment*, 539-541, 1972.

11. Schecter, A. J. "The inpatient unit reconsidered," *Proceedings, National Conference on Methadone Treatment*, 1:476-481, 1973.

12. Schecter, A. J., J. Friedman, and D. Grossman. "Clinical use of naltrexone (EN-1639A). Part I: Efficacy and safety in pilot studies," *Amer. J. Drug and Alcohol Abuse*, I:2, 253-269, 1974.

13. Kreek, M. J., A. J. Schecter, C. L. Gutjahr, D. Bowen, F. Field, J. Queenan and I. Merkatz. "Methadone levels in maternal and neonatal body

fluids during maintenance treatment, *Amer. J. Drug and Alcohol Abuse*, I:3, 409-418, 1974.

14. Schecter, A. J. "Consumer acceptance of drug abuse programs: A provider's view," *J. Psychedelic Drugs*, 6:2, 213-223, 1974.

15. Schecter, A. J. and D. Grossman. "Naltrexone in a clinical setting: Preliminary observations," *Proceedings, National Academy of Sciences Committee on Problems of Drug Dependence*, 264-279, 1974.

16. Schecter, A. J. and F. Kauders. "Methadone and L-alpha-acetylmethadol in a treatment program in Brooklyn," *Amer. J. Drug and Alcohol Abuse*, II:¾, 331-339, 1975.

17. Schecter, A. J. "Clinical use of naltrexone (EN-1639A). Part II: Experience with the first 50 patients in a New York treatment clinic," *Amer. J. Drug and Alcohol Abuse*, II:¾, 433-442, 1975.

18. Schecter, A. J. and F. Kauders. "Patient deaths in a narcotic antagonistic (Naltrexone) and L-alpha-acetylmethadol program," *Amer. J. Drug and Alcohol Abuse*, II:¾, 443-449, 1975.

19. Schecter, A. J. and D. Grossman. "Experience with Naltrexone: A suggested role in drug abuse treatment programs," Developments in the Field of Drug Abuse, *Proceedings, 1974 of the National Association for the Prevention of Addiction to Narcotics*, Senay, E., Shorty, V., and Alksne, H. (eds.). Cambridge, Massachusetts: Schenkman Publishing Company, Inc., 754-766, 1975.

20. Schecter, A. J. (Abstract) "The career teacher training program for medical school faculty members and its impact on the training of American physicians in drug and alcohol abuse," In: *Developments in the Field of Drug Abuse*, Proceedings, 1974 of the National Association for the Prevention of Addiction to Narcotics, Senay, E., Shorty, V., Alksne, H. (eds.). Cambridge, Massachusetts:

Schenkman Publishing Company, Inc., 1110-1111, 1975.

21. Schecter, A. J. "Naltrexone: Observations with fifty patients during 14 months," *Proceedings, National Academy of Science, Committee on Problems of Drug Dependence*, 533-542, 1975.
22. Schecter, A. J. "Preface," *Rehabilitation Aspects of Drug Dependence*, Schecter, A. (ed.). Cleveland, Ohio: CRC Press, 1977.
23. Schecter, A., "An overview of the use of narcotic antagonists in the rehabilitation of opiate dependent persons," *Critical Concerns in the Field of Drug Abuse, Proceedings of the Third National Drug Abuse Conference*, Inc., New York, 1976, Schecter, A., Alksne, H. and Kaufman, E., (eds.) New York, New York: Marcel Dekker, Inc., 1260-1268, 1978.
24. Schecter, A., "Historical perspectives on the use of levo-alpha-acetylmethadol (LAAM or LAM) in the rehabilitating opiate dependent persons, "*Critical Concerns in the Field of Drug Abuse*, Proceedings of the Third National Drug Abuse Conference, Inc., New York, 1976, Schecter, A., Alksne, H. and Kaufman, E., (eds.) New York, New York: Marcel Dekker, Inc., 1317-1321, 1978.
25. Schecter, A. J. and V. Shorty "Preface," *Drug Abuse: Modern Trends, Issues, and Perspectives*, Proceedings of the Second National Drug Abuse Conference, Inc., New Orleans, Louisiana, 1975, Schecter, A., Alksne, H. and Kaufman, E. (eds.). New York, New York: Marcel Dekker, Inc., 1978.
26. Schecter, A. and F. Kauders. "Naltrexone in a clinical setting the first 50 patients," *Drug Abuse: Modern Trends, Issues, and Perspectives*, Proceedings of the Second National Drug Abuse Conference, Inc., New Orleans, Louisiana, 1975, Schecter, A.J., Kaufman, E., and Alksne, H. (eds.). New York, New York: Marcel Dekker, Inc., 508-516, 1978.

27. Schecter, A. and F. Kauders. "Methadone and long-acting methodone in a New York drug abuse treatment program" *Drug Abuse: Modern Trends, Issues, and Perspectives*, Proceedings of the Second National Drug Abuse Conference, Inc., New Orleans, Louisiana, 1975, Schecter, A., Alksne, H., and Kaufman, E. (eds.). New York, New York: Marcel Dekker, Inc., 540-557, 1978.
28. Schecter, A. and D. Grossman. "Medical and public health school curriculum in drug and alcohol abuse: a national survey, *"Drug Abuse Modern Trends, Issues, and Perspectives*, Proceedings of the Second National Drug Abuse Conference, Inc., New Orleans, Louisiana, 1975, Schecter, A.J., Alkesne H., and Kaufman, E. (eds.). New York, New York: Marcel Dekker, Inc., 985-993, 1978.
29. Wolfson, E. and A. Schecter. "A unique approach to drug dependence in the medical school curriculum," A>Multicultural View of Drug Abuse, *Proceedings of the National Conference on Drug Abuse*, Cambridge, Massachusetts: Schenkman Publishing Co., 263-271, 1978.
30. Schecter, A. "Preface," *Treatment Aspects of Drug Dependence*, Schecter, A. (ed.). West Palm Beach, Florida: CRC Press, 1978.
31. Schecter, A. and M. J. Schecter. "The role of long-acting methadone (LAAM) in the treatment of opiate dependence," *Treatment Aspects of Drug Dependence*, Schecter, A. (ed.). West Palm Beach, Florida: CRC Press, Inc., 33-40, 1978.
32. Schecter A. and M. J. Schecter. "Naltrexone in the rehabilitation of opiate addicts," *Treatment Aspects of Drug Dependence*, Schecter, A. (ed.) West Palm Beach, Florida: CRC Press, 59-70, 1978.
33. Marshall, C., J. Salzer, A. Memon and A. Schecter. "An obligatory undergraduate emergency medicine course," *J. Med. Educ.*, 54:652-566, 1979.

34. Schecter, A. "The role of narcotic antagonists in the rehabilitation of opiate addicts: a review of naltrexone," *Amer. J. Drug Alcohol Abuse,* 7(1): 1-18, 1980.

35. Kreek, M. J., A. J. Schecter, C. L. Gutjahr, and M. Hecht. "Methadone use in patients with chronic renal disease," *Drug and Alcohol Dependence,* 5: 197-205, 1980.

36. Schecter, A.J. and J.M. Last. "Substance abuse: Drug dependence and alcoholism," *Maxcy-Rosenau's Public Health and Preventative Medicine,* 11th Edition, Last, J. (ed.). New York, New York: Appleton-Century-Crofts, 1077-1094, 1980.

37. Schecter, A. "Long acting methadone (Levo-alpha-acetylmethadol) in the treatment of opiate dependence," *Yearbook of Drug Use and Abuse,* Brill, L. and Winnick, C. (eds.). Human Sciences Press, 99-112, 1980.

38. Schecter, A. "A new sociocultural-clinical approach to the use of narcotic antagonists in rehabilitation of opiate dependent persons," *Drug Dependence and Alcoholism,* Vol. I, Biomedical Issues, Schecter, A. (ed.). New York, New York: Plenum Press, 973-980, 1981.

39. Schecter, A. "Now that LAAM's here, is take home methadone desirable?," *Drug Dependence and Alcoholism, Vol. I: Biomedical Issues,* Schecter, A. (ed.). New York, New York: Plenum Press, 1051-1055, 1981.

40. Schecter, A., G. Haughie and R. Rothenberg "PCB Transformer Fire—Binghamton, New York," *Morbidity and Mortality Weekly Report,* 30:16, 187-193, 1981.

41. Loekle, D. M., A. J. Schecter and J.J. Christian. "Effects of chloroform, tetrachloro-ethylene and trichloroethylene on survival, growth and liver of

poecillia sphenops," *Bulletin Environm. Contam. Toxicol.,* 30:199-205, 1983.

42. Shecter, A. "Contamination of an office building in Binghamton, New York by PCB's, dioxins, furans and biphenylenes after an electrical panel and electrical transformer incident," *Chemosphere,* 12:4/5, 669-680, 1983.

43. Schecter, A. "Light and ultrastructural characteristics of neuroblastoma glioma hybrid NG 108-15 cells," *Life Sciences,* 33:719-722, 1983.

44. Schecter, A., F. Schaffner, T. Tiernan and M. Taylor. "Ultrastructural alterations of liver mitochondria in response to dioxins, furans, PCB's and biphenylenes," *Banbury Report 18: Biological Mechanisms of Dioxin Action,* Cold Spring Harbor Laboratory: Banbury Center Publications, 177-190, 1984.

45. Tiernan, T. O., M. L. Taylor, G. F. VanNess, J. H. Garrett, S. R. Bultman, C. B. Everson, J D. Hinders, A. J. Schecter. "Analyses of human tissues for chlorinated dibenzo-p-dioxins and chlorinated dibenzofurans: the current state of the art," *Public Health Risks of the Dioxins: Proceedings of a Rockefeller University Symposium,* Lowrance, W.W. (ed.). Los Altos, California: Kaufman Publishing Co., 31-56, 1984.

46. Ryan, J. J., A. Schecter, R. Lizotte, W. F. Sun and L. Miller. "Tissue distribution of dioxins and furans in humans from the general population," *Chemosphere,* 14:6/7, 929-932, 1985.

47. Schecter, A., J. J. Ryan, R. Lizotte, W. F. Sun, L. Miller, G. Gitlitz and M. Bogdasarian. "Chlorinated dibenzodioxin and dibenzofurans in human adipose tissue from exposed and control New York State patients," *Chemosphere,* 14:6/7, 933-937, 1985.

48. Schecter, A., T. A. Gasiewicz, H. Eisen and F. Schaffner. "Ultrastructural alterations in liver cells of humans, rats and mouse hepatoma cells in response to 2,3,7,8-tetrachlorodibenzo-p-dioxin and related compounds," *Chemosphere*, 14:6/7, 939-944, 1985.

49. Schecter, A., T. Tiernan, M. Taylor, G. F. VanNess, J. H. Garrett, D. J. Wagel, G. Gitlitz and M. Bogdasarian. "Biological markers after exposure to polychlorinated dibenzo-p-dioxins, dibenzofurans, biphenyls and biphenylenes. Part I: Findings using fat biopsies to estimate exposure," *Chlorinated Dioxins and Dibenzofurans in the Total Environment II*, Keith, L., Rappe, C., and Choudhary, G. (eds.). Stoneham, Massachusetts: Ann Arbor Science, Butterworth Publishers, 215-245, 1985.

50. Schecter, A., F. Schaffner, T. Tiernan, M. Taylor, G. F. VanNess, J. H. Garrett, D.J. Wagel, G. Gitlitz, and M. Bogdasarian. "Biomedical markers after exposure to polychlorinated dibenzo-p-dioxins, dibenzofurans, biphenyls and related chemicals. Part II: Ultrastructural characterization of human liver biopsies," *Chlorinated Dioxins and Dibenzofurans in the Total Environment*, II, Keith, H., Rappe, C., and Choudhary, G. (eds.). Stoneham, Massachusetts: Ann Arbor Science, Butterworth Publishers, 247-265, 1985.

51. Tiernan, T. O., M. L. Taylor, J. H. Garrett, G. F. VanNess, J. G. Solch, D. J. Wagel, G. L. Ferguson and A. Schecter. "Sources and fate of polychlorinated dibenzodioxins, dibenzofurans and related compounds in human environments," *Environmental Health Perspectives*, 59:145-158, 1985.

52. Schecter, A., T. Tiernan, F. Schaffner, M. Taylor, G. Gitlitz, G. F. VanNess, J.H. Garrett and D.J. Wagel. "Patient fat biopsies for chemical analysis

and liver biopsies for ultrastructural characterization after exposure to polychlorinated dioxins, furans and PCBs," *Environmental Health Perspectives*, 60:241-254, 1985.

53. Schecter, A. and T. Tiernan. "Occupational exposure to polychlorinated dioxins, polychlorinated furans, polychlorinated biphenyls and biphenylenes after an electrical panel and transformer accident in an office building in Binghamton, NY," *Environmental Health Perspectives*, 60:305-313, 1985.

54. Schecter, A. "Medical surveillance of exposed persons after exposure to PCB's, chlorinated dibenzodioxins, and dibenzofurans after PCB transformer or capacitor incidents," *Environmental Health Perspectives*, 60:333-338, 1985.

55. Schecter, A. "Toxicology," *McGraw-Hill Yearbook of Science and Technology 1986*, New York, New York: McGraw-Hill Publishing Co., 457-460 1985.

56. Schecter, A., Contributor to "Bioavailability of Dioxins," *U.S. Environmental Protection Agency 1984 Workshop Proceedings*, Courtney, D. (ed.). Government Printing Office.

57. Huang Lu, C. J., D. Redmond, R. Baggs, A. Schecter and T. A. Gasiewicz. "Growth and hepatic composition in the guinea pig after long-term parenteral hyperalimentation," *American Journal of Physiology*, 251:388-397, 1986.

58. Schecter, A. "The Binghamton state office building PCB, dioxin and dibenzofuran electrical transformer incident; 1981-1986," *Chemosphere*, 15:9/12, 1273,1280, 1986.

59. Schecter A., J. J. Ryan and J. D. Constable. "Chlorinated dibenzo-p-dioxin and dibenzofurans levels in human adipose tissue and milk samples from the

north and south of Vietnam," *Chemosphere*, 15:9/12, 1613-1620, 1986.

60. Gasiewicz, T. A., E. C. Henry, R. Baggs, Rucci, and A. Schecter. "Temporal and dose-related characteristics of biochemical and morphological alterations in the hamster induced by 2,3,7,8-tetrachlorodibenzo-p-dioxin," *Chemosphere*, 15:9/12, 1749-1752, 1986.

61. Weerasinghe, N. C. A., A. J. Schecter, J. C. Pan, R. L. Lapp, D. E. Giblin, J. L. Meehan, L. Hardell and M. L. Gross. 'Levels of 2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD) in adipose tissue of U.S. Vietnam veterans seeking medical assistance," *Chemosphere*, 15:9/12 1787-1794, 1986.

62. Eisen, H. J., A. Schecter, T. Cresteil, A. K. Jaiswal. "Actions of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCCD) in the human heptoblastoma $HepG_2$ tissue culture cell Line; functional and electron microscopic observation," *Chemosphere*, 15:9/12, 1965-1970, 1986.

63. Ryan, J. J., A. Schecter, W. F. Sun and R. Lizotte. "Distribution of chlorinated dibenzo-p-dioxins and chlorinated dibenzofurans in human tissues from the general population," *Chlorinated Dioxins and Dibenzofurans in Perspectives*, Rappe, C., Choudhary, G., Keith, L., (eds.), Chelsea, Michigan: Lewis Publishing Co., 1:3-16, 1986.

64. Schecter, A. J., J.J. Ryan, M. Gross, N. C. A. Weerasinghe and J. D. Constable. "Chlorinated dioxins and dibenzofurans in human tissues from Vietnam, 1983-1984," *Chlorinated Dioxins and Dibenzofurans in Perspective*, Rappe, C., Choudhary, G., and Keith, L. H. (eds), Chelsea, Michigan: Lewis Publishing Co., 3:35-50, 1986.

65. Schecter, A., J. J. Ryan and G. Gitlitz. "Chlorinated dioxin and dibenzofuran levels in human adipose tissues from exposed and control populations," *Chlorinated Dioxins and Dibenzofurans in Perspective*, Rappe, C., Choudhary, G., and Keith, L, (eds.), Chelsea, Michigan: Lewis Publishers, 4:51-65, 1986.

66. Schecter, A. "Dioxin Toxicity" *The McGraw-Hill Encyclopedia of Science and Technology*, 6th Edition, 1987, New York, New York: McGraw-Hill Publishing Co., 1986.

67. Huang Lu, C. J., R. Gabbs, D. Redmond, E. Henry, A. Schecter, T. Gasiewicz. "Toxicity and evidence for metabolic alterations in 2,3,7,8-Tetrachlorodibenzo-p-dioxin treated guinea pigs fed by total parenteral nutrition," *Toxicology and Applied Pharmacology*, 84:439-453, 1986.

68. Schecter, A. "Transient liver pathology in patients consuming water from a private well contaminated by PCB's from a submersible water pump," *Chemosphere*, 16:1 37-42, 1987.

69. Schecter, A., H. Eichelberger and H. Eisen. "Transmission and scanning electron microscopic characterization of mouse HEPA 1 hepatoma cells after 2,3,7,8-tetrachloro-para-dibenzodioxin treatment," *Chemosphere*, 16:8/9, 1713-1718, 1987.

70. Schecter, A., J. D. Constable, S. Arghestani, H. Tong and M. L. Gross. "Elevated levels of 2,3,7,8-tetrachlorodibenzodioxin in adipose tissue of certain U.S. veterans of the Vietnam war," *Chemosphere*, 16:8/9, 1997-2002, 1987.

71. Schecter, A., J. J. Ryan and J. Constable. "Polychlorinated dibenzo-p-dioxin and polychlorinated dibenzofuran levels in human breast milk from Vietnam compared with cow's milk and human breast

milk from the North American continent," *Chemosphere*, 16:8/9, 2003-2016, 1987.

72. Ryan, J. J., A. Schecter, Y. Masuda and M. Kikuchi. "Comparison of PCDDs and PCDFs in the tissues of Yusho patients with those from the general population in Japan and China," *Chemosphere*, 16: 8/9, 2017-2025, 1987.

73. Constable, J. D. and A. Schecter. "Human fat sampling for dioxin analysis," *Chemosphere*, 16:8/9, 2063-2068, 1987.

74. Schecter, A. and T. Gasiewicz. "Health hazard assessment of chlorinated dioxins and dizenzofurans contained in human milk," *Chemosphere*, 16:8/9, 2147-2154, 1987.

75. Schecter, A. "The Binghamton state office building PCB transformer incident: 1981-1987," *Chemosphere*, 16:8/9, 2155-2160, 1987.

76. Schecter, A. " Risk assessment and standard setting, rapporteur's summary and discussion," *Chemosphere*, 16:8/9, 2205-2210, 1987.

77. Schecter, A. J., M. Gross and J. J. Ryan. "Biological markers of exposure to chlorinated dioxins and dibenzofurans in the United States and Vietnam," *Hazardous Materials Disposal: Sitting and Management*, Manas Chatterji (ed.), Aldershot, England and Brookfield, VT: Gower Publishers, Ltd., 79-90, 1987.

78. Schecter, A. and R. Stephens. "A comparison of electrical transformer accidents in Binghamton and San Francisco," *Hazardous Materials Disposal: Siting and Management*, Manas Chatterji (ed.), Aldershot, England and Brookfield, VT: Gower Publishers, Ltd., 147-165, 1987.

79. Schecter, A. and T. A. Gasiewicz. "Human breast milk levels of dioxins and dibenzofurans and their

significance with respect to current risk assessments," Solving Hazardous Waste Problems: Learning from Dioxins, Jurgin H. Exner (ed.), *American Chemical Society Symposium Series No. 191*, American Chemical Society, Washington, D.C. 162-173, 1987.

80. Schecter, A. and E. W. Cassell. "Substance use and abuse," *The Preventive Approach to Patient Care*, Sheridan, D.P. and Winogrond, L.R., (eds[.]), Elsevier Science Publishing Co. Inc., 12:310-334, 1987.

81. Schecter, A., A. Dekin, N. C. A. Weerasinghe, S. Arghestani and M.L. Gross. "Sources of dioxins in the environment: a study of PCDDs and PCDFs in ancient, frozen Eskimo tissue," *Chemosphere*, 17:4, 627-631, 1988.

82. Schecter, A., J. J. Ryan. "Polychlorinated dibenzo-para-dioxin and dibenzofuran levels in human adipose tissues from workers 32 years after occupational exposure to 2,3,7,8-TCDD," *Chemosphere*, 17:5, 915-920, 1988.

83. Schecter, A., J. D. Constable, J. V. Bangert, H. Tong, S. Arghestani, S. Monson and M. Gross. "Elevated body burdens of 2,3,7,8-tetrachlorodibenzodioxin in adipose tissue of United States Vietnam veterans," *Chemosphere*, 18:1/6, 431-438, 1989.

84. Schecter, A., P. Fürst, C. Kruger, H. A. Meemken, W. Groebel and J. D. Constable. "Levels of polychlorinated dibenzofurans, dibenzodioxins, PCBs, DDT and DDE, hexachlorobenzene, dieldrin, hexachlorocyclo- hexanes and oxychlordane in human breast milk from the United States, Thailand, Vietnam, and Germany," *Chemosphere*, 18: 1/6, 445-454, 1989.

85. Schecter, A., J. D. Constable, J. V. Bangert, J. Wiberg, M. Hansson, M. Nygren and C. Rappe. "Isomer specific measurement of polychlorinated dibenzodioxin and dibenzofuran isomers in human blood from American Vietnam veterans two decades after exposure to Agent Orange," *Chemosphere*, 18: 1/6, 531-538, 1989.

86. Schecter, A., R. Kooke, P. Serné, K. Olie, J. Constable. "Chlorinated dioxin and dibenzofuran levels in food samples collected between 1985-87 in the north and south of Vietnam," *Chemosphere*, 18: 1/6, 627-634, 1989.

87. Schecter, A. and J. J. Ryan. "Blood and adipose tissue levels of PCDDs/PCDFs over three years in a patient after exposure to polychlorinated dioxins and dibenzofurans," *Chemosphere*, 18:1/6, 635-642, 1989.

88. Schecter, A., J. Mes and D. Davies. "Polychlorinated biphenyl (PCB), DDT, DDE and hexachlorobenzene (HCB) and PCDD/F isomer levels in various organs in autopsy tissue from North American patients," *Chemosphere*, 18:1/6, 811-818, 1989.

89. Schecter, A., B. D. Eitzer and R. A. Hites. "Chlorinated dioxin and dibenzofuran levels in sediments collected from rivers in Vietnam, 1984-6," *Chemosphere*, 18:1/6, 831-834, 1989.

90. Fürst, P., Chr. Kruger, H. A. Meemken, W. Groebel and A. Schecter. "Interaction between sample and packaging material—a potential source of contamination with PCDDs and PCDFs," *Chemosphere*, 18:1/6, 891-896, 1989.

91. Schecter, A., J. J. Ryan and J. D. Constable. "Chlorinated dioxins and dibenzofurans in human milk from Japan, India, and the United States of America," *Chemosphere*, 18:1/6, 975-980, 1989.

92. Schecter, A., H. Y. Tong, S. J. Monson, M. L. Gross and J. Constable. "Adipose Tissue Levels of 2,3,7,8-TCDD in Vietnamese adults living in Vietnam, 1984-87," *Chemosphere*, 18:1/6, 1057-1062, 1989.

93. Nguyen Thi Ngoc Phuong, Bui Sy Hung, Dan Quoc Vu and Arnold Schecter. "Dioxin levels in adipose tissues of hospitalized women living in the south of Vietnam in 1984-85 with a brief review of their clinical histories," *Chemosphere*, 19:1/6, 933-936, 1989.

94. Pham Hoang Phiat, Trinh Kim Anh, Dan Quoc Vu and Arnold Schecter. "Preliminary observations on the clinical histories, polychlorinated dibenzodioxin and dibenzofuran tissue levels and 2,3,7,8-TCDD toxic equivalents of potentially dioxin exposed patients living in the south of Vietnam," *Chemosphere*, 19:1/6, 937-940, 1989[.]

95. Schecter A., P. Fürst, C. Fürst, H. A. Meemken, W. Groebel, and Dan Quoc Vu. "Levels of polychlorinated dibenzodioxins and dibenzofurans in cows's milk and in soy bean derived infant formulas sold in the United States and other countries," *Chemosphere*, 19:1/6, 913-918, 1989.

96. Schecter A., P. Fürst, J. J. Ryan, C. Fürst, H. A. Meemken, W. Groebel, J. Constable, and Dan Vu. "Polychlorinated dioxin and dibenzofuran levels from human milk from several locations in the United States, Germany and Vietnam," *Chemosphere*, 19:1/6, 979-984, 1989.

97. Schecter A., D. Vu, H. Y. Tong, S. J. Monson, M. L. Gross and J. D. Constable. "Levels of 2,3,7,8-TCDD and 2,3,7,8-TCDF in human adipose tissue from hospitalized persons in the north and south of Vietnam 1984-88," *Chemosphere*, 19:1/6, 1001-1004, 1989.

98. Schecter A., H. Y. Tong, S. J. Monson and M. L. Gross. "Levels of 2,3,7,8-TCDD in silt samples collected between 1985-86 from rivers in the north and south of Vietnam," *Chemosphere*, 19:1/6, 547-550, 1989.

99. Olie, K., A. Schecter, J. Constable, R. M. M. Kooke, P. Serné, P. C. Slot and P. de Vries. "Chlorinated dioxin and dibenzofuran levels in food and wildlife samples in the north and south of Vietnam," *Chemosphere*, 19:1/6, 493-496, 1989.

100. Nguyen Thi Ngoc Phuong, Bui Sy Hung, A. J. Schecter, and Dan Quoc Vu. "Dioxin levels in adipose tissues of hospitalized women living in the south of Vietnam in 1984-89 with a brief review of their clinical histories," *Women & Health*, Vol. 16 (1) 1990.

101. Schecter A., H. Y. Tong, S. L. Monson, M. L. Gross, S. Raisanen, T. Karhunen, E. K. Osterklund, J. D. Constable, Hoang Dinh Cau, Le Cao Dai, Hoang Tri Quynh, Ton Duc Lang, Nguyen Thi Ngoc Phoung, Pham Hoang Phiet, and D. Vu. "Human adipose tissue dioxin and dibenzofuran levels and 'Dioxin Toxic Equivalents' in patients from the north and south of Vietnam," *Chemosphere*, 20:7/9, 943-950, 1990.

102. Schecter A., J. J. Ryan, J. D. Constable, R. Baughman, J. Bangert, P. Fürst, K. Wilmers, and R. P. Oates. "Partitioning of 2,3,7,8-chlorinated dibenzo-p-dioxins and dibenzofurans between adipose tissue and plasma lipid of 20 Massachusetts Vietnam Veterans," *Chemosphere*, 20:1/7, 951-958, 1990.

103. Schecter A., J. R. Startin, M. Rose, C. Wright, I. Parker, D. Woods, and H. Hansen. "Chlorinated dioxin and dibenzofuran levels in human milk from Africa, Pakistan, southern Vietnam, the southern USA and England," *Chemosphere*, 20:7/9, 919-925, 1990.

104. Schecter A., P. Fürst, C. Fürst, W. Groebel, J. D. Constable, S. Kolesnikov, A. Beim, A. Boldonov, E. Trubitsun, B. Vlasov, Hoang Dinh Cau Dai, and Hoang Tri Quynh. "Levels of Chlorinated dioxins, dibenzofurans, and other chlorinated xenobiotics in food from the Soviet Union and the south of Vietnam," *Chemosphere*, 20:7/9, 799-806, 1990.

105. Schecter A., P. Fürst, C. Fürst, W. Groebel, S. Kolesnikov, M. Savchenkov, Beim A., Boldonov A., Trubitsun E., and Vlasov B. "Levels of dioxins, dibenzofurans, and other chlorinated xenobiotics in human milk from the Soviet Union," *Chemosphere*, 20:7/9, 927-934, 1990.

106. Olson J.R., B. P. McGarrigle, D. A. Tonucci, A. Schecter, and H. Eichelberger. "Developmental toxicity of 2,3,7,8-TCDD in the rat and hamster," *Chemosphere*, 20:7/9, 1117-1123, 1990.

107. Tong H. Y., M. L. Gross, A. Schecter, S. J. Monson, and A. Dekin. "Sources of dioxins in the environment: Second stage study of PCDD/Fs in ancient human tissue and environmental samples," *Chemosphere*, 20:7/9, 987-992, 1990.

108. Schecter A., J. J. Ryan, and P. Kostyniak. "Decrease over a six year period of dioxin and dibenzofuran tissue levels in a single patient following exposure," *Chemosphere*, 20:7/9, 911-917, 1990.

109. Schecter A., P. Fürst, C. Fürst, and W. Groebel. "Human milk dioxin and dibenzofuran levels and levels of other chlorinated chemicals from various countries, including Vietnam and Cambodia, as compared to the Soviet Union. In: *Orango-Halogen Compounds*, Eco-Informa Press, Bayreuth, Germany. Vol. 1:267, 1990.

110. Schecter, A., H. Eichelberger, and J. Olsen, "Light and electron microscopy observations in kidney of maternal and fetal hamster after dosing with 2,3,7, 8-TCDD." In: *Organo-Halogen Compounds*, Eco-Informa Press, Bayreuth, Germany. Vol. 1:207, 1990.

111. Schecter, A., O. Päpke, and M. Ball. "Chlorinated dioxin and dibenzofuran levels in human blood samples from various countries, including Vietnam, the Soviet Union, the United States and Germany." In: *Orgando-Halogen Compounds*, Eco-Informa Press, Bayreuth, Germany. Vol. 1:271, 1990.

112. Schecter, A., J. J. Ryan, and T.A. Gasiewicz. "Decrease in human tissue levels of dioxins and dibenzofurans over nine years after exposure in one male patient." In: *Organo-Halogen Compounds*, Eco-Informa Press, Bayreuth, Germany. Vol. 1:275, 1990.

113. Schecter, A., J. J. Ryan, O. Päpke, and M. Ball. "Comparison of dioxin and dibenzofuran levels in whole blood, blood plasma and adipose tissue, on a lipid basis." In: *Organo-Halogen Compounds*, Eco-Informa Press, Bayreuth, Germany. Vol. 1: 279, 1990.

114. Wilson-Yang, K.M., E. S. Chisholm, D. Hallett, J. P. Poer, and A. Schecter. "The congener specific determination of PCBs: analyses of whole blood." In: *Organo-Halogen Compounds*, Eco-Informa Press, Bayreuth, Germany, Vol. 2:241, 1990.

115. Schecter, A. "Problems encountered in cleanup of a PCB, dibenzofuran, and dioxin contaminated building." In: *Organo-Halogen Compounds*, Eco-Informa Press, Bayreuth, Germany. Vol. 3:409, 1990.

116. Schecter A., O. Päpke, and M. Ball. "Evidence for transplacental transfer of dioxins from mother to fetus: Chlorinated dioxin and dibenzofuran levels in the livers of stillborn infants," *Chemosphere* 21: 8, 1017-1022, 1990.

117. Schecter A. "The Massachusetts Agent Orange Study," *Agent Orange: Medical Scientific, Legal, Political and Psychological Issues*, P. Atwood, ed. University of Massachusetts, Boston: Joiner Center for the Study of War and Social Consequences, 1990.

118. Schecter A., "Fallout from Agent Orange" In the Letters column, *Time* 08/20, 1990.

119. Schecter A., T. Gasiewicz, T. Guidotti, D. Phillips, J. J. Ryan, C. Burrell, S. Conibear, and H. Hanson "Clinical medicine of exposure to toxic chemicals," in W. Flint and J. Vena, eds. *Human Health Risks from Chemical Exposure: The Great Lakes Ecosystem*. Chelsea, Mich.: Lewis Publishers, 1991.

120. Schecter, A., and J. J. Ryan. "Chlorinated and brominated dioxin levels in the blood of a chemist who became ill after synthesizing 2,3,7,8-TCDD and 2,3,7,8-TBrDD." In: *Organo-Halogen Compounds*, Eco-Informa Press, Bayreuth, Germany. Vol. 4: 141, 1991.

121. Schecter, A., J. J. Ryan, A. Di Domenico, F. Merli, V. Diaco, and A. Gallo. "Dioxin and dibenzofuran levels in adipose tissue from an office worker working near the Seveso, Italy dioxin incident." In: *Organo-Halogen Compounds*, Eco-Informa Press, Bayreuth, Germany. Vol. 4:145, 1991.

122. Wolff, M., and A. Schecter. "Accidental exposure of children to polychlorinated biphenyls." *Archives Environmental Contamination and Toxicology* 20:4, 449, 1991.

123. Schecter, A. "Dioxins and related chemicals in humans and the environment." In: *Banbury Report 35: Biological Basis for Risk Assessment of Dioxins and Related Compounds*. Cold Spring Habor Laboratory, Cold Spring Harbor, New York, 169-213, 1991.

124. Schecter, A., B. J. Poiesz, P. W. Brandt-Rauf, O. Päpke, and M. Ball. "Dioxin levels in [redacted] of AIDS patients and controls." *Medical Science Research*, 19:9, 273-275, 1991.

125. Schecter, A., R. Malkin, O. Päpke, M. Ball, and P. W. Brandt-Rauf[.] "Dioxin levels in blood of municipal incinerator workers." *Medical Science Research*, 19:11, 331-332, 1991.

126. Schecter A. and J. J. Ryan. Brominated and chlorinated dioxin blood levels in a chemist 34 years after exposure to 2,3,7,8-tetrachlorodibenzodioxin and 2,3,7,8-tetrabromodibenzodioxin. *Chemosphere* 23: 11/12, 1921-1924, 1991.

127. Schecter A., P. Fürst, C. Fürst and O. Päpke. Dioxin, dibenzofurans and chlorinated organic compounds in human milk and blood from Cambodia, Germany, Thailand, U.S.A., U.S.S.R. and Vietnam. *Chemosphere* 23:11/12, 1903-1912, 1991.

128. Schecter A. and K. Charles. The Binghamton State Office Building transformer fire incident after one decade. *Chemosphere* 23:8/10, 1307-1321, 1991.

129. Schecter A., O. Päpke[,] M. Ball and J. J. Ryan. Partitioning of dioxins and dibenzofurans: whole blood, blood plasma and adipose tissue. *Chemosphere* 23:11/12, 1913-1919, 1991.

130. Schecter, A., H. McGee, J. Stanley and K. Boggess. "Dioxin, Dibenzofuran, and PCB levels in the blood of Vietnam veterans in the Michigan Agent Orange Study," *Chemosphere*, 25:1/2, 205-208, 1992.

131. Schecter, A., H. McGee, J. Stanley and K. Boggess. "Chlorinated Dioxin, Dibenzofuran and coplanar PCB levels in blood and semen of Michigan Vietnam veterans," *Organohalogen Compounds*: Extended Abstracts-Dioxin '92, 9:231-234, 1992.

132. Schecter, A., O. Päpke, M. Ball, Hoang Dinh Cau, Le Cao Dai, Hoang Trong Quynh, Ding Quang Minh, Nguyen Ngoc Thi Phuong, Huynh Kim Chi, and Pham Hoang Phiet. "The mapping out of environmental levels of dioxin contamination by use of pooled whole blood throughout a country: Vietnam, a case study," *Organohalogen Compounds*: Extended Abstracts-Dioxin '92, 9:235-238, 1992.

133. Schecter, A., O. Päpke, M. Ball and Y. Masuda. "Distribution of dioxins and dibenzofurans in blood from Japan, Israel, Russia, Guam, Vietnam, Germany, and the U.S.A.," *Organohalogen Comp onds*: Extended Abstracts-Dioxin '92, 9:239-242, 1992.

134. Schecter, A., O. Päpke, M. Ball, J.R. Startin, C. Wright and M. Kelly. "Dioxin and dibenzofuran levels in food from the United States as compared to levels in food from other industrial countries" *Organohalogen Compounds*: Extended Abstracts-Dioxin '92, 9:243-246, 1992.

135. Schecter, A., J.J. Ryan, O. Päpke, M. Ball and V. Zheleznyak. "Dioxin and dibenzofuran levels in the blood of exposed male and female Russian workers with chloracne as compared to controls," *Organohalogen Compounds*: Extended Abstracts-Dioxin '92, 9:247-250, 1992.

136. Schecter, A., J.R. Startin, C. Wright, M. Kelly, G. Lucier and K. Charles. "Dioxin and dibenzofuran levels in Yucheng placentas and control placentas comparing dioxin/dibenzofuran levels with receptor

binding and enzyme induction," *Organohalogen Compounds*: Extended Abstracts-Dioxin '92, 9: 251-254, 1992.

137. Lambert, G.H., D.A. Scholler, C.C. Hsu, P. Taylor, H.E.B. Humphrey, F. Garcia, L. Dai, M. Budd, A. Schecter, and H. Lietz. "Cytochrome P4501A2 activity in humans exposed to PCBs and dioxins," *Organohalogen Compounds*: Extended Abstracts-Dioxin '92, 10:139-142, 1992.

138. Schecter, A., O. Päpke, M. Ball, B. Poiesz and P.W. Brandt-Rauf. "A followup study of dioxin levels in the blood of AIDS patients with opportunistic infections as compared to HIV positive asymptomatic persons and controls," *Organohalogen Compounds*: Extended Abstracts-Dioxin '92, 10:205-207, 1992.

139. Schecter, A. A. di Domenico L. Turrio-Baldassarri and J.J. Ryan. "Dioxin and dibenzofuran levels in the milk of women from four geographical regions in Italy as compared to levels in other countries," *Organohalogen Compounds*: Extended Abstracts-Dioxin '92, 10:227-230.

140. Schecter, A. and J.J. Ryan. "Persistent brominated and chlorinated dioxin blood levels in a chemist 35 years after dioxin exposure," *Journal of Occupational Medicine*, 34:7, 702-707, 1992.

141. Schecter, A., O. Päpke, M. Ball, M. Grachev, A. Beim, V. Koptug, Hoang Dinh Cau, Le Cao Dai, Hoang Tri Quynh, Nguyen Ngoc Thi Phuong, Huynh Kim Chi. "Dioxin and dibenzofuran levels in human blood samples from Guam, Russia, Germany, Vietnam and the U.S.A.," *Chemosphere*, 25:7/10, 1129-1134, 1992.

142. Schecter, A., O. Päpke, M. Ball, Hoang Dinh Cau, Le Cao Dai, Nguyen Quang Ming, Hoang Trong Quynh, Nguyen Ngoc Thi Phuong, Pham Hoang

Phiet, Huynh Kim Chi, Dieu Thieu Vo, J.D. Constable, and J. Spencer. "Dioxin and dibenzofuran levels in blood and adipose tissue of Vietnamese from various locations in Vietnam in proximity to Agent Orange spraying," *Chemosphere*, 25:7/10, 1123-1128, 1992.

143. Schecter, A. "Dioxins and dibenzofurans in exposed workers; serial tissue levels in a worker exposed in a PCB transformer fire cleanup and blood levels in three exposed chemists," *Chemosphere*, 25:7/10, 1117-1122, 1992.

144. Schecter, A., J. J. Ryan and J. D. Constable. "Partitioning of dioxin and dibenzofuran congeners between plasma and cell fractions of blood from 10 adult male patients," *Chemosphere*, 25:12, 2017-2022, 1992.

145. Schecter, A., P. Fürst, C. Fürst, M. Grachev, A. Beim and V. Koptug. "Levels of dioxins, dibenzofurans and selected other chlorinated organic compounds in food from Russia." *Chemosphere* 25:12, 2009-2015, 1992.

146. Wolff, M. and A. Schecter. "Use of PCB levels to assess potential exposure following an electrical transformer explosion," *Journal of Occupational Medicine*, 34:11, 1079-1083, 1992.

147. Schecter, A., J. Stanley, K. Boggess, Y. Masuda, J. Mes, M. Wolff, P. Fürst, C. Fürst, H. McGee, K. Wilson-Yang, and B. Chisholm. "Polychlorinated biphenyl levels in the tisues of exposed and non-exposed humans," in press, *Environmental Health Perspectives*, 1992.

148. Schecter, A., J. J. Ryan, O. Päpke, M. Ball, Y. Masuda, P. Brandt-Rauf, J. Constable, Hoang Dinh Cau, Le Cao Dai, Hoang Tri Quynh, Nguyen Thi Ngoc Phuong, and Pham Hoang Phiet. "Chlorinated

and brominated dioxins and dibenzofurans in human tissue following exposure," in press *Environmental Health Perspectives*, 1992.

149. Schecter, A., P. Fürst, C. Fürst, O. Päpke, M. Ball, J.J. Ryan, Hoang Dinh Cau, Hoang Trong Quynh, H.Q. Cuong, Nguyen Thi Ngoc Phuong, Pham Hoang Phiet, A. Beim, M. Grachev, J. Constable, J. Startin, M. Samedy, and Yit Kim Seng. "Chlorinated dioxins and dibenzofurans in human tissues from general populations: A selective review," in press, *Environmental Health Perspectives*, 1992.

150. Wilson-Yang, K.M., J.P. Power, E.A. Chisholm, D.J. Hallett and A. Schecter. "The analysis of human blood for seventy-five PCB congeners by dual column capillary-gas chromatography," *Chemosphere*, 25:7/10, 1375-1379, 1992.

151. Schecter, A., Ryan, J.J., Päpke, O., Ball, M., and Lis A. "Elevated Dioxin Levels in the Blood of Male and Female Russian Workers with and without Chloracne 25 Years After Phenoxyherbicide Exposure: The Ufa 'Khimprom' Incident," in press *Chemosphere*, 1993.

152. Schecter, A., McGee, H., Stanley, J., Boggess, K. "Chlorinated dioxin, dibenzofuran, coplanar, mono-ortho, and di-ortho substituted PCB congener levels in blood and semen of Michigan Vietnam veterans compared with levels in Vietnamese exposed to Agent Orange. in press *Chemosphere*, 1993.

IN THE UNITED STATES DISTRICT COURT
FOR THE NORTHERN DISTRICT OF GEORGIA
ATLANTA DIVISION

(Title Omitted in Printing)

AFFIDAVIT OF ARTHUR L. FRANK, MD, Ph.D.

Personally appeared before me, the undersigned officer duly authorized to administer oaths, Arthur L. Frank, MD, Ph.D., who after being duly sworn, deposes as follows:

1.

My name is Arthur L. Frank, MD, Ph.D. I have reached my majority and am mentally competent. I have personal knowledge of the facts set forth in this affidavit.

2.

I reside at 3320 Gondola Court, Lexington, Kentucky, 40513. My professional address is University of Kentucky, Department of Preventive Medicine and Environmental Health, Chandler Medical Center, Lexington, Kentucky, 40536-0084.

3.

I am a Professor and Chairman of the Department of Preventive Medicine and Environmental Health at the University of Kentucky College of Medicine. I am also the Director of the Occupational Medicine Program and Director of Graduate Studies, Master of Science in Public Health Degree Program.

I am a specialist in the field of occupational medicine, toxicology and preventive medicine. I obtained my medical degree from the Mount Sinai School of Medicine of the City of New York in 1972 and my Ph.D. from the City University of New York, Biomedical Sciences Pro-

gram at the Mount Sinai Medical Center in 1977. A true and current copy of my curriculum vitae is attached to this affidavit as Exhibit "A". It accurately reflects my training and experience. Exhibit "B" to this affidavit accurately reflects my publications and presentations in the field relevant to the opinions given in this affidavit.

4.

I have written numerous peer reviewed papers, and have written papers reviewing the works of others, in the field of the epdimology and etiology of lung cancer. I have been interested in the subject of occupational lung cancer for much of my academic career and have researched this field extensively. My papers concerning occupational lung cancer, and the epidemiology and etiology of lung cancer, have been supported, in many cases, by original research that I conducted and directed. I have been qualified as an expert witness to assist juries in understanding the causes of lung cancer on numerous occasions, and in numerous courts, throughout the United States.

5.

Lung cancer, like other cancers, begins with an initiated cell. The initiator can be any number of things including, but not limited to, tobacco smoke. An initiated cell does no harm by itself until it is promoted. An initiated cell can survive for a number of years without any harmful effects on the body. There are a wide variety of known and suspected promoters of lung cancer.

6.

Epidemiology studies and statistics, and my own personal observations and experience, indicate that lung cancer is extremely rare for a thirty seven year old white male in the United States. This is true even for persons with a history of tobacco use. Lung cancer, as a general rule, is not seen until much later in life.

7.

Based on available studies, and my person observations and experience, it is my opinion, to a reasonable degree of medical certainty, that lung cancer in a thirty-seven (37) year old white male, even with a history of cigarette smoking, would be as a result of exposure to other exogenous factors which lead to the rapid development of the malignancy. The exogenous factors are often referred to as promoters and are what make, or promote, an essentially harmless initiated cell into a harmful malignant cancer.

8.

I am familiar through my training and experience and research, with the types of cancer which attack the lung. I have reviewed the medical records of Robert Joiner with respect to his lung cancer and have reviewed his use of tobacco and exposure to tobacco smoke. The cell type of cancer that Mr. Joiner has, the type known as "small cell" carcinoma of the lung, has multiple recognized causes, including, but not limited to, tobacco smoke and such occupational exposures as ionizing radiation and exposure to bischloromethyl ether. Other occupational carcinogens, likewise have been associated with the development of this type of carcinoma. No cancer is exclusively one type of cell. No one cell type is exclusively related to cigarette smoke or any other single causative agent. In other words, the fact that Mr. Joiner has a small cell type of lung cancer does not, in and of itself, provide any definitive link to tobacco smoke being the cause of that cancer. It is more likely than not, given Mr. Joiner's limited tobacco use, and also considering his second hand tobacco smoke exposure, and given his age at the onset of lung cancer, 37 years, that tobacco smoke served only as the initiator of the cancer and that some other agent served as the promotor of the initiated cells. It was the promotion of the initiated cells which caused Mr. Joiner to be harmed.

By: /s/ Arthur L. Frank, MD
ARTHUR L. FRANK, MD, Ph.D.

Subscribed and sworn before me on this 28th day of January, 1994.

/s/ Rebecca L. Payne
Notary Public
My commission expires: 1-13-98

IN THE UNITED STATES DISTRICT COURT
FOR THE NORTHERN DISTRICT OF GEORGIA
ATLANTA DIVISION

(Title Omitted in Printing)

AFFIDAVIT OF DANIEL T. TEITELBAUM, M.D.

Daniel T. Teitelbaum, M.D., being duly sworn, deposes and says:

1. My name is Daniel T. Teitelbaum, M.D.; I have personal knowledge of every statement made in this Affidavit.

I am a practicing medical toxicologist in Denver, Colorado. I am adjunct professor of Environmental Science and Engineering Ecology at the Colorado School of Mines, where I teach the graduate toxicology courses in occupational and environmental toxicology. I am also associate clinical professor of medicine and preventive medicine at the University of Colorado Health Sciences Center, where I teach toxicology and the epidemiology of toxic diseases. In addition, I am visiting professor of medicine and toxicology at the faculty of medicine of Technion, the Israel Institute of Science, in Haifa, Israel. I am a founder and former director of Rocky Mountain Poison Control Center, and of the Physicians Poison Consultation Service at the University of Colorado Medical Center. I have been chairman of the Diagnostic Product Advisory Committee and the Toxicology Diagnostic Product Advisory Committee of the United States Food and Drug Administration. I have been a member of several committees and consultation panels of the United States Environmental Protection Agency. I was a member of a Blue Ribbon Panel of the Executive Office of the Presi-

dent which was called together to advise on future direction of research for the Environmental Protection Agency and the National Institutes of Environmental Health Sciences. I have been a special consultant for the United States Occupational Safety and Health Administration on many occasions. I assisted in the development of standards for workplace exposure to benzene, formaldehyde, ethylene dibromide and other materials. I participated in the development of the medical surveillance protocols for workers exposed to lead, formaldehyde and benzene, and I testified at the OSHA public hearings dealing with these Standards.

In the recent past, I have taught two courses at the Federal Judicial Center under the auspices of George Washington University for the Federal Judiciary on issues of toxicology and the epidemiology of toxic diseases.

I have written more than forty articles which are published in peer reviewed journals. I have also presented many lectures and talks in the area of toxicology and the diagnosis and management of toxic disease.

I have been an advisor and panel member of several World Health Organization working parties on toxic disease, including a new panel on genetic susceptibility to toxic substances.

I received my M.D. degree in 1964 from the Albert Einstein College of Medicine and I completed an internship at Montefiore Hospital in New York City. I was a resident in internal medicine and a fellow in toxicology at the University of Colorado Health Sciences Center. I completed my formal training in 1968.

I was a founder of the American Academy of Clinical Toxicology and the American Board of Medical Toxicology. I was first chairman of the examining board of the American Board of Medical Toxicology. I was certified by the American Board of Toxicology in 1975 and recertified by examination in 1976.

I am currently involved in the day to day practice of medical toxicology and in consultation in the area of medical toxicology, for medical legal purposes, for corporate clients, for labor, for government agencies, regulatory agencies, police agencies and for civil litigation. In the course of my work I have interviewed, examined and treated numerous patients who were employed in the electrical trades. These tradesmen and craftsmen included licensed electricians, electronic technicians and technologists, engineers, linemen, and powerhouse employees. In addition, I have interviewed and examined transformer repairmen for utility companies, private manufacturers, govenment agencies, and railroad and other transportation companies. I have visited powerhouses, transmission substations, field transmission sites, electrical and electronic manufacturing and assembly plants and other facilities in which the technical and craft persons whom I interviewed and examined were employed.

As a result of this work, I have extensive knowledge of the handling of toxic substances, hazardous materials, electrical power, and other risks of a physical and chemical nature which are common to the electrical crafts and trades.

A copy of my curriculum vitae is attached as Exhibit 1.

2. During my evaluation of the matter of Joiner v. Westinghouse, et al., I carried out a comprehensive and traditional occupational medical assessment of Mr. Joiner in the offices of Northside Internists and Endocrinologists in Atlanta, Georgia. As part of this assessment I interviewed and examined him in Atlanta, Georgia on July 14, 1993, for several hours. In addition, I reviewed his past medical records, the data which was available about his workplace and materials with which he worked, depositions of Mr. Joiner, and others, and depositions of family members and co-workers about the nature of his work.

I also considered many other documents relevant to the questions which I was asked concerning Mr. Joiner's illness and its relationship to his occupational exposures to toxic substances.

In the course of my toxicologic evaluation of Mr. Joiner I utilized traditional medical assessment techniques. I also relied upon my extensive experience with workers in the electrical trades and my knowledge of the toxicology of the materials with which Mr. Joiner worked. I considered the fundamental mechanisms of toxicology and carcinogenesis as a manifestation of toxic outcome, the biology of cancer including the biology of small cell lung cancer, and the state of the art regarding the testing and evaluation of toxic substances for carcinogenic risk in humans. Attached to this affidavit as Exhibit 2 is a list of documents which I have reviewed in connection with this case. These documents have previously been furnished to defendants in the course of my deposition which was taken in Jacksonville, Florida on October 12, 1993.

MR. JOINER'S MEDICAL HISTORY

1. In the course of my evaluation of Mr. Joiner's current clinical condition and in order to understand the genesis of his lung cancer within the context of contemporary thought on carcinogenesis, I took an extensive medical history from Mr. Joiner. This medical history was provided to the defendants as an exhibit to my deposition (Dr. Teitelbaum's Deposition, Exhibit 10.) His medical history revealed there were a number of risk factors for lung cancer present in Mr. Joiner. These factors were, first, a family history of lung cancer in his mother, who died at age 64 and was a smoker. There is a less clear history of cancer in other relatives, which I could not further classify. A second risk factor was the fact his father had worked at a sand and gravel

mining company until the patient was approximately 7 or 8 years old. There is no evidence that Mr. Joiner has silicosis or any scarring as a result of household contact with silica which his father might have brought home on his clothing. However, current thought suggests that silica is a lung carcinogen in humans. It is possible but not at all probable that domestic exposure to silica may have contributed to Mr. Robert Joiner's overall lung cancer risk. Third, Mr. Joiner was a cigarette smoker from age 19 to age 27. According to the history which he gave to me and the statement in his deposition, he smoked 1 package of cigarettes per 24 hours for eight years, which yields an 8 pack year dose. He stopped smoking completely at age 27. Mr. Joiner avoided all cigarette smoke after the fourth Friday of April, 1982. Fourth, in 1973 Mr. Joiner began to work for the Thomasville Electric Utility, as a transformer repairman. He worked on oil filled transformers, many of which were contaminated with polychlorinated biphenyl.

According to the extensive occupational history which I took from Mr. Joiner and according to the documents provided to me, his deposition, and my knowledge of the usual practice of electrical transformer repairmen in the era in question, it is clear that Mr. Joiner worked with PCB contaminated mineral oil, mineral oil per se, mineral spirits as a cleaning agent, and paints. He was repeatedly exposed to the inhalation of smoke and fume from the process of "baking out transformers" utilizing stadium lights as a heat source. His work practices clearly indicated to me that in accord with well established principles of pharmacokinetics he would have been substantially exposed to polychlorinated biphenyls and polychlorinated dibenzofurans in mineral oil, mineral oil per se, and mineral spirits, by skin, respiratory and oral route and to fumes, smoke, dust and aerosol from "baking out of transformers" during the course of his work. I did not

find any other risk factors for lung cancer or any other significant exposures in his occupational history.

MR. JOINER'S CIGARETTE SMOKING AND IT'S RELATIONSHIP TO THE CAUSATION OF HIS LUNG CANCER

1. In the defendants' motion for summary judgement, defendants' attorneys propose that "even as Mr. Joiner's own experts acknowledge he has had consistent, substantial exposure to cigarette smoke for many years." This statement could not be farther from the truth. The defendants' attorneys cite my deposition at pages 48, 54, 55 and 56, both out of context and incorrectly. On page 48, lines 1-11, I stated as follows, "Mr. Joiner smoked for a relatively significant period of his life, but from the point of view of causation of lung cancer, a very—a low-end dose and early, and he stopped 12 years approximately before he got sick. But if the most recent studies on smoking and cancer are correct, previous assumptions that as time passed the risk would disappear have been unduly optimistic. So we have some risk of lung cancer because he was a smoker; however, the likelihood of his developing lung cancer at age 37 on a statistical basis is extremely small." I then continued, lines 12-17, "If you compared the lung cancer rates at age 60, which are around 400 per 100,000 person years, with lung cancer rates in the 30's, which are almost too low to detect, somewhere around 10 to 15 per 100,000 and most of those in the later 30's rather than the early 30's, it's pretty—pretty low risk, but it's there." By no stretch of the imagination can this comment be interpreted to mean what defendants have suggested I said on page 9 of their motion for summary judgement. Consideration of the further references on pages 54, 55 & 56 of my deposition will confirm that at no point did I concede or would I have agreed that Mr. Joiner had "consistent, substantial exposure to cigarette smoke for many years." Mr. Joiner's

smoking history has been related above. It is contained in the medical history which I took and is restated in his deposition. While there may be some discrepant smoking history in his medical record, that history was not given under oath. His history under oath in his deposition and my medical history are concordant.

2. A remote smoking history of 8 pack years can only be considered a minimal risk factor for the causation of lung cancer. Such a history cannot be associated with any particular cell type of lung cancer. So few lung cancers at this age have been collected in the literature that no statistically meaningful analysis of histological type at age 37 is possible. On the basis of the data which is available, it is clearly extraordinary for lung cancer to develop in a 37 year old male with a brief smoking history which terminated eleven years prior to the onset of his cancer. The age and risk factor considerations strongly point to the operation of other carcinogens and cocarcinogens in Mr. Joiner's cancer, since cigarette smoking is so unlikely to be a sufficient cause of lung cancer at his age.

3. In their motion for summary judgment, defendants' attorneys suggest that some special relationship exists between small cell cancer of the lung and cigarette smoking. They do not cite any particular authority for this position. Based upon my knowledge and understanding of the literature dealing with lung cancer and cigarette smoking it is apparent that they are quoting a paper by Stayner and Wegman[1] which was published in 1983. The report suggested that, "cigarette smoking was significantly associated with all three histological types of lung cancer, with the strongest relationship being for

---

[1] Stayner LT; Wegman DH
Smoking, occupation and histopathology of lung cancer: a case-control study with the use of the Third National cancer survey. JNCI 1982; 70:421-6

small cell carcinoma."[2] Further study since 1983 has clearly established that the relationship between cigarette smoking and lung cancer is very strong, and that the most likely type of cancer to develop is squamous cell carcinoma, not small cell cancer. This likelihood that squamous cell cancer will develop in white male smokers is present at virtually all ages. Small cell cancer represents only 14 to 15 percent of all cancers in cigarette smokers who are white males at all ages.[3] Stockwell, et al., in a very large study of 35,000 smoking lung cancer deaths in Florida reported that "Among males, adenocarcinomas represent the most frequent cell type only among younger, never-smoking males. In other categories squamous carcinomas dominate."[3]

4. The Stockwell study and other studies have confirmed that the current state of the art is as described by Hegmann, et al., 1993, that "There was no predisposition toward a specific histologic type of lung cancer" in cigarette smokers.[3] The mass of literature on smoking and lung cancer makes it clear that there is no particular predisposition to any given histologic type of cancer among smokers. It also makes it manifest that the occurrence of lung cancer in a smoker whose dose of cigarettes is as low as Mr. Joiner's and who is as young as Mr. Joiner, is very unusual.

5. Small cell lung cancer has been reported in radiation exposed individuals,[4] in radon exposed individu-

---

[2] Stockwell HG; Armstrong AW; Leaverton PE
Histopathology of lung cancers among smokers and nonsmokers in Florida.
Int J Epidemiol 1990; 19 Suppl 1:S48-52

[3] Hegmann KT; Fraser AM; Keaney RP; Moser SE; Nilasena DS; Sedlars M; Higham-Gren L; Lyon JL
The effect of age at smoking initiation on lung cancer risk.
Epidemiology 1993 Sep;4(5):444-8

[4] Land CE; Shimosato Y; Saccomanno G; Tokuoka S; Auerbach O; Tateishi R; Greenberg SD; Nambu S; Carter D; Akiba S; et al

als,[5] in persons exposed to chloromethyl ethers,[6] and in other toxic exposures[7] which have resulted in lung cancer. In my opinion, the extraordinary occurrence of lung cancer, at a young age and with minimal cigarette smoking in a person whose latency period is brief, and whose family history of cigarette smoking and lung cancer does not provide an adequate explanation for the occurrence of this disease, demands further analysis. I have carried out such an analysis, in accord with accepted toxicologic practice, and I have concluded the principal additional risk factors which are present and which have determined the causation of lung cancer in Mr. Joiner in a multifactorial fashion were: 1. work with PCB contaminated mineral oils, mineral spirits, and mineral oil per se; 2. his family history, and; 3. his smoking history. Together these factors have combined to result in lung cancer at a very young age in a minimal smoker. I believe that the scientific literature strongly supports this position.

Radiation-associated lung cancer: a comparison of the histology of lung cancers in uranium miners and survivors of the atomic bombings of Hiroshima and Nagasaki.
Radiat Res 1993 May;134(2):284-43

[5] Biberman R; Lusky A; Schlesinger T; Margaloit M; Neeman E; Modan B
Increased risk for small cell lung cancer following residential exposure to low-dose radon: a pilot study.
Arch Environ Health 1993 Jul-Aug;48(4):209-12

[6] Figueroa WG; Raszkowski R; Weiss W
Lung cancer in chloromethyl methyl ether workers.
N Engl J Med 1973 May 24;288(21):1096-7

[7] Ishikawa Y; Mori T; Kato Y; Tsuchiya E; Machinami R; Sugano H; Kitagawa T
Lung cancers associated with thorotrast exposure: high incidence of small-cell carcinoma and implications for estimation of radon risk.
Int J Cancer 1992 Oct 21:52(4):570-4

MR. JOINER'S WORK WITH PCB CONTAINING OIL AND HIS EXPOSURE TO PCBs

1. The defendants' attorneys in their motion for summary judgement have called into question my assessment of the extent and significance of Mr. Joiner's exposure to polychlorinated biphenyls in the mineral oil of the transformers with which he worked. They concede that at least 20 percent of the transformers handled at the Thomasville utility facility were contaminated with polychlorinated biphenyls manufactured by their clients. However, they argue that this exposure could not have produced a dose which was sufficient to cause Mr. Joiner's cancer. They incorrectly allege that I did not adequately consider the information available concerning the PCB contamination of the mineral oils in the transformers which Mr. Joiner handled.

2. During the course of my deposition I indicated that I had relied upon certain documents furnished by plaintiff's attorneys concerning the level of PCB contamination in various transformers. These documents included specific incident reports, during which Mr. Joiner was directly involved in the salvage of PCB containing transformers which had been involved in a lightning strike. The transformers were contaminated with PCBs up to levels of several hundred parts per million (Dr. Teitelbaum's deposition, Exhibit 16-B). In addition, a lightning strike and overheating of a transformer in the presence of oxygen in the dielectric fluid, inevitably produces polychlorinated dibenzofurans. PCDFs are carcinogens which are generally believed to contaminate all transformers at varying concentrations when PCBs, oxygen, and electric current combine. Such situations occur during arcing and transformer fires. Thus, Mr. Joiner would also have been exposed to polychlorinated dibenzofurans.

At the time of my deposition, I had not been furnished with specific transformer data concerning PCB content

which was then being analyzed by the plaintiff's attorneys. Since that time, I have received a database printout of analyses of transformer PCB content based upon documents furnished to the plaintiffs by the defendants. These documents indicated that 50.8 percent of the transformers handled by the Thomasville utility contained PCB's in concentrations greater than zero. Twenty-one percent of the transformers demonstrated PCB contents of 0 to 50 parts per million, 25 percent demonstrated concentrations of 50 to 500 parts per million, 4 percent contained greater than 500 parts PCB per million. Thus, 29.3 percent of all of the transformer fluids analyzed contained polychlorinated biphenyls at greater than 50 parts per million, and 21.5% had levels greater than zero but less than 50 ppm. Since transformers may contain between eight and five hundred gallons of transformer oil, the potential and actual exposure to Mr. Joiner would be enormous. (Personal Communication, Public Service Co., Colorado, 1994) [8] In my opinion, this is significant contamination and is consistent with what has been described in the scientific literature. It represents a substantial source of exposure to polychlorinated biphenyls for Mr. Joiner and his co-workers.

3. How polychlorinated biphenyls which are present in transformer fiuid are absorbed into the human body to deliver a dose of toxic substance to the target organs of the worker has been well defined. In Mr. Joiner's case it is clear that at least 2 significant routes and one minor route of exposure were present. The first of these routes was inhalation of mist and fume. The second was dermal exposure, and the third minor route was oral exposure.

Mr. Joiner reported that he constantly inhaled oil fumes. A standard working practice at the Thomasville utility included the use of stadium lamps to dry the cores of

[8] Personal communication, Public Service Company of Colorado, Jan. 1994

transformers being repaired in order to rehabilitate them. This drying process, which had to be carried out at a temperature above 180 degrees F. in order to achieve significant evaporation of the oil put a substantial amount of contaminated fume in the air. This resulted in eye, airway and chest irritation in Mr. Joiner. It delivered directly to the target organ, Mr. Joiner's lung, a significant dose of mineral oil, polychlorinated biphenyls, furans, mineral spirit and other contaminants.

The practice of heating PCB containing transformer parts to dry them out is not new. The disingenuous implication in the memorandum of the defendants' attorneys in their motion for summary judgment that there is something odd, peculiar or non-standard about this practice of heating transformer cores and interior parts in order to achieve oil drying, is surprising. In Hubert's text, entitled "Preventive Maintenance of Electrical Equipment," second edition, published by McGraw-Hill Book Company in 1969, there is a description of the process of heating mineral oil and Askarel® containing transformers in order to achieve drying. A specific warning appears on Page 73 as follows, "Warning. Every effort should be made to avoid getting liquid Askarel® or concentrated vapors from hot Askarel® on the skin. It is very irritating, especially to the eyes, nose and lips. Continued exposure to Askarel® may cause skin eruptions because of the absorption of the liquid through the pores of the skin. The recommended treatment for such irritations is castor oil for the eyes, and castor oil or cold cream for the nose, lips and other areas of skin."[9] Clearly it was well known by industrial engineers as long ago as 1969, that heating of PCB containing mineral oils in transformers could lead to chloracne and was a cause of substantial absorption of PCBs. Other studies have confirmed that this is so.

---

[9] Hubert CI
Preventive Maintenance of Electrical Equipment.
2nd edition. New York: McGraw-Hill, 1969.

4. In an artfully crafted question which appears on page 58, lines 6,7 and 8 of my deposition, defendants' attorneys asked me, "Do you know what the skin absorption rate through the skin is of PCBs that are contained in contaminated mineral oil?" In response to that question, I indicated that I could not find any specific data on the question of rate of absorption. Studies had been done prior to the time of my deposition which address the issue of absorption in animal skin. There were no specific studies which dealt with living human skin. In 1990, Wester et al.,[10] published a paper entitled, "Percutaneous absorption and skin decontamination of PCBs: in vitro studies with human skin and in vivo studies in the rhesus monkey" in the Journal of Toxicology and Environmental Health; the investigators demonstrated that PCBs are efficiently absorbed through the skin, and that excretion from the body is slow. The vehicle within which the PCBs which were contained, water, trichlorobenzene, or mineral oil did not affect percutaneous absorption. In vitro skin absorption in human cadaver skin did not correlate with in vivo findings. This was due to the lack of PCB partitioning from the skin into the water receptor fluid, even with the addition of 6 percent Oleth 20® solubilizer. Since 1990, the same group of investigators has published more on the skin absorption of PCB. Wester, et al., in another paper in the Journal of Toxicology and Environmental Health, 1993, entitled, "Percutaneous absorption of PCBs from soil: In vivo rhesus monkey, in vitro human skin, and binding to powdered human stratum corneum" demonstrated that, "With in vitro percutaneous absorption through human skin, most of the Aroclor 1242 and Aroclor 1254 resided in the skin and the amounts

---

[10] Wester RC; Maibach HI; Bucks DA; McMaster J; Mobayen M; Sarason R; Moore A
Percutaneous absorption and skin decontamination of PCBs: in vitro studies with human skin and in vivo studies in the rhesus monkey.
J Toxicol Environ Health 1990 Dec;31(4):235-46

were dependent upon dosing vehicle (water > mineral oil > soil)." [11] They further indicated that in the rhesus monkey, the percutaneous absorption of Aroclor 1242 was 13.8, plus or minus 2.7 percent of the dose. The absorption of Aroclor 1254 was 14.1, plus or minus 1 percent of the dose. These absorption amounts were similar to the absorption of Aroclor 1242 and 1254 from other vehicles such as mineral oil, trichlorobenzene, acetone. According to Wester's work, it is likely that humans absorb significant percentages of an administered dose of PCBs from a mineral oil vehicle, such as that to which Mr. Joiner was exposed. That the specific study has not been done in living human beings is hardly surprising. The general awareness of the extreme toxicity of the PCBs and their co-riders in the mineral oil would make it impossible to carry out an absorption study under the terms of current human investigational protocols.

5. Dermal absorption as a major route of body entry for PCBs has been addressed effectively in the medical literature. In a paper published in the American Industrial Hygiene Association Journal, 1987, Peter S.J. Lees and colleagues reported, "Evidence is presented to support the hypothesis that the dermal and dermal/oral routes of worker PCB exposure are the major contributors to total PCB body burden in the group of transformer maintenance and repair personnel studied." [12] Their calculations demonstrate that Mr. Joiner was significantly exposed to PCBs in mineral oil.

---

[11] Wester RC; Maibach HI; Sedik L; Melendres J; Wade M
Percutaneous absorption of PCBs from soil: in vivo rhesus monkey, in vitro human skin, and binding to powdered human stratum corneum.
J Toxicol Environ Health 1993 Jul;39(3):375-82

[12] Lees PS; Corn M; Breysse PN
Evidence for dermal absorption as the major route of body entry during exposure of transformer maintenance and repairmen to PCBs.
Am Ind Hyg Assoc J 1987 Mar;48(3):257-64

This paper also confirmed that while the respiratory route could be a meaningful source of exposure to PCBs, "dermal contact and absorption of PCBs is the most significant source of exposure and the resultant body burden." [13] It is clear that Mr. Joiner, handled transformer oil without protective gear on many days during the course of his employment at the Thomasville utility facility, repeatedly entered into large transformers, transferred large quantities of transformer oil, handled the oil, kept his tools oiled with the oil, and had extensive dermal oil contact. In addition, Mr. Joiner had substantial respiratory exposure to oils, PCB and PCDF, because of the deliberate heating of transformer cores in order to dry them. His exposure was intensified by exposure to the heated transformer oil which was present as a result of arcing, lightning strikes, fires and other inevitable mishaps in the operation of transformers in the field. Together, these resulted in a significant, repeated acute and chronic exposure to polychlorinated biphenyls, polychlorinated dibenzofurans, mineral oil, mineral spirits and other contaminants within that mineral oil. I believe that it is more probable than not that Mr. Joiner underwent a significant, substantial, meaningful, toxic, and carcinogenic exposure to these materials between 1973 when he began to work at Thomasville and the time that his cancer occurred.

ROLE OF ANIMAL STUDIES IN PREDICTING HUMAN CARCINOGENICITY

1. Defendants' attorneys have suggested that animal studies are unreliable as a basis of opinions as to causation of cancer in humans. In my opinion, defendants have selectively read the literature. They continue to discount the enormous contributions made to the prediction of human cancer risk by carefully done animal studies. In a 1993 review of chronic life span toxicology studies, Monro commented as follows, "For such compounds, a study in rats at pharmacodynamically relevant dose levels

for 12-18 months will, in general, provide adequate information for carcinogenic risk assessment in humans." [13] Others, including Huff, in his most recent paper in The Journal of Pharmacology and Toxicology, entitled, "Issues and controversies surrounding qualitative strategies for identifying and forecasting cancer causing agents in the human environment" commented that, "Long term laboratory animals bioassays, despite the criticism leveled at them, were considered to be the most useful and valid techniques for identifying potential human carcinogens." [14] Defendants' dismissal of animal studies which have consistently found PCBs to be carcinogenic at all levels of testing is merely a reflection of their desire to dodge the bullet on this issue.

EVIDENCE SUPPORTING THE POSITION THAT PCBS ARE CARCINOGENIC IN HUMANS AND ARE A CONTRIBUTING CAUSE TO THE OCCURRENCE OF HUMAN LUNG CANCER

1. The defendants' attorneys argue that there is insufficient scientific evidence to conclude that polychlorinated biphenyls are carcinogenic. They do not believe that it is more probable than not that PCB and oil and PCDFs and mineral spirits contributed to Mr. Joiner's lung cancer. In my opinion, lung cancer is a probable toxic outcome of exposure to polychlorinated biphenyls. I believe that there is scientific evidence which supports the proposition that polychlorinated biphenyls are carcinogenic, are

---

[13] Monro A

How useful are chronic (life-span) toxicology studies in rodents in identifying pharmaceuticals that pose a carcinogenic risk to humans?

Adverse Drug React Toxicol Rev 1993 Spring;12(1):5-34

[14] Huff J

Issues and controversies surrounding qualitative strategies for identifying and forecasting cancer causing agents in the human environment.

Pharmacol Toxicol 1993;72 Suppl 1:12-27

carcinogenic in man, and are able to produce lung cancer in humans.

2. The most recent conclusions of the International Programme on Chemical Safety of the World Health Organization regarding polychlorinated biphenyls are stated in Environmental Health Criteria, No. 140, published in 1993.[15] In this document, the WHO confirms that, "The respiratory tract is certainly the most important route of exposure in the case of acute emergency situations, where unprotected personnel working in areas containing such PCB concentrations may, in theory, inhale a total dose of up to 10 mg/day." They further point out that, "transient irritation of the mucous membranes of the respiratory tract has been reported in emergency situations, as well as difficulty in breathing at high concentrations." They continue on Page 470 with the following statement, "The situation is totally different in the case of acute exposures to heated or decomposed PCBs, in which the inhaled total concentrations might be several orders of magnitude higher than above, though the irritative effect may prevent breathing in such contaminated rooms. Since the soot often contains a considerable fraction of particles, a few microns in size, it is partly breathed in and, thus, may lead to alveolar retention of both soot and absorbed chemicals." [16] The Environmental Health Criteria Document (EHCD) describes precisely the type of exposure which Mr. Joiner underwent. This respiratory exposure, in addition to his dermal exposure, provides further support for the delivery of the PCBs to the target organ, his lung.

In the EHCD, the editors note the significance of a mortality study of PCB workers carried out at the Mon-

---

[15] IPCS—International Programme on Chemical Safety Environmental Health Criteria 140: Polychlorinated Biphenyls and Terphenlys.

2nd edition. Geneva: World Health Organization, 1993.

santo plant in Sauget, Illinois by Zack and Musch[16] in 1979. The preliminary report of this study appears in discovery documents provided in connection with this case under the Bates number JOI003183 and following. This epidemiologic study was never published. However, it is cited in the WHO document. Zack and Musch found a general increase in malignancies in the exposed group, 8 observed verses 4.4 expected for an SMR of 179. Lung cancer was elevated, with 4 observed versus 1.44 expected. Although adequate adjustment for confounding because of exposure to additional materials in the workplace was not done, workers who utilized materials quite like those Mr. Joiner used had an elevated incidence of lung cancer.

3. In another study performed by the defendants' in this case in 1975, which they did not furnish to the World Health Organization IPCS, but also appears in the discovery documents associated with this case commencing at Bates number JOI003180, Dr. Mahboubi and his colleagues at the Eppley Institute of Cancer at the University of Nebraska carried out a life table analysis of a group of employees of the Monsanto Krummrich plant, at which PCBs were manufactured.[17] This study which defendants have not published and which is not generally available in the literature but was discovered in the course of this lawsuit, demonstrated, according to Fredrich R. Johansen, staff toxicologist at Monsanto, that, "When compared to the 1969 U.S. male population, this group of 23 deaths would be expected to contain 1.24 cases attributable to lung cancer. Statistical comparison of the observed lung cancer verses the expected lung cancer yielded a highly significant chi x^2 value of 7.1. This number is even more

[16] Zack JA; Musch DC
Mortality of PCB workers at the Monsanto Plant in Sauget, Illinois. Monsanto Chemical Co., St. Louis, MO. 1979. (unpublished report)

[17] Mahboubi E
Correspondence and tables sent to Frederick R. Johannsen, Ph.D., Toxicologist.
Monsanto Company, St. Louis, MO. October 17, 1975.

significant than the value (chi $x^2 = 6.8$) obtained following evaluation of the full 50-person mortality population." [18] Again, defendants' own epidemiologic studies confirm that lung cancer appears in excess in PCB exposed workers.

4. Bertazzi, et al., in an ongoing study of PCB exposed capacitor workers, first published in 1982 and updated several times since, has noted that total cancer deaths were elevated, (12 observed versus 5.3 expected) and that hematological neoplasms were also elevated, (4 observed versus 1.1 expected).[19] Lung cancer was also increased, although, the findings at the time of the last report did not reach statistical significance. Since the cohort under observation has not reached full maturity, it is likely that additional lung cancer cases will occur and that statistical significance will develop in this group of PCB exposed workers also.[19]

5. Observations of Yusho patients, Japanese patients who suffered the adverse effects of consumption of PCB contaminated rice oil, showed elevated SMR's for all malignant neoplasms, including stomach cancers, liver cancers and lung cancers. Statistically significant excess mortality was seen for cancer of the lung.[20]

6. A study of considerable importance to Mr. Joiner's case appeared in the British Journal of Industrial Medicine in 1988, in two parts. These papers by Ronneberg

[18] Johannsen FR
Memo to G. Roush, Monsanto, re: Mahboubi report and tables. Monsanto, Dept. Medicine & Environmental Health, St. Louis, MO. August 27, 1976.

[19] Bertazzi PA; Riboldi L; Pesatori A; Radice L; Zocchetti C
Cancer mortality of capacitor manufacturing workers.
Am J Ind Med 1987;11:165-76

[20] Ikeda M; Kuratsune M; Nakamura Y; Hirohata T
A cohort study on mortality of yusho patients—a preliminary report.
Fukuoka Igaku Zasshi 1987 May;78(5):297-300

and colleagues,[21,22] investigated the mortality and incidence of cancer among oil exposed workers in a Norwegian cable manufacturing company. In Part 2, the mortality and cancer incidence study between 1953-84, a statistically significant excess of deaths from lung cancer in transformer oil exposed workers was observed. Ten deaths from lung cancer were observed where 3.9 were expected. Nine of the cases of lung cancer occurred 20 or more years after first employment, where 2.7 would have been expected yielding a P value of 0.01, a highly significant result. In the subcohort of workers who had at least one year's employment in oil exposed work, there were 7.06 cases of lung cancer per 1,000 person years in smokers compared to 1.30 cases of lung cancer in smokers in the general population sample. It appears that exposure to transformer oil and low viscosity mineral oils were superadditive or perhaps, synergistic carcinogens in these workers. Transformer oils were utilized in impregnation and sheathing from 1940 to 1945 and in installation from 1941 to 1970.

Review of these four studies strongly supports my contention that PCBs do cause lung cancer in humans. In my opinion, PCBs were a significant contributing cause to Mr. Joiner's lung cancer.

MULTI-FACTORIAL CAUSATION OF CANCER

1. Defendants' have suggested that my formulation of the basis for causation of Mr. Joiner's cancer is defective

---

[21] Ronneberg A; Skyberg K

Mortality and incidence of cancer among oil exposed workers in a Norwegian cable manufacturing company. Part I. Exposure conditions 1920-79.

Br J Ind Med 1988 Sep;45(9):589-94

[22] Ronneberg A; Andersen A; Skyberg K

Mortality and incidence of cancer among oil exposed workers in a Norwegian cable manufacturing company. Part 2. Mortality and cancer incidence 1953-84.

Br J Ind Med 1988 Sep;45(9):595-601

because it relies upon a "mix" theory. This pejorative term is applied by defendants' attorneys in order to support their belief that plaintiffs experts in general and I in particular should point to a single cause for Mr. Joiner's cancer. Their position flies in the face of scientific knowledge. It defies the current state of the art regarding causation of cancer, and approaches a "junk science" theory.

2. It is uniformly believed by responsible scientists that cancer is a multifactorial and multi-stage process. The exact mechanism by which any type of cancer is caused is still only partially known, but many steps have been defined. This understanding forms the basis for my opinion that Mr. Joiner's cancer is a multifactorial disease, to which PCBs, smoking, family history and other materials contributed.

3. Vainio and Wilbourn in Pharmacology and Toxicology, 1993, indicate that cancer is the result of "both endogenous and environmental factors, ranging from exposure to a single identified chemical to the occupations we follow in order to make our living."[23]

4. Burnstein, in surgical clinics of North America, 1993, indicated that the etiology of colorectal cancer is multifactorial.[24]

5. Correa, in Cancer Research, 1992, indicated that, human gastric cancer is multistep and multifactorial process.[25]

---

[23] Vainio H; Wilbourn J

Cancer etiology: agents causally associated with human cancer. Pharmacol Toxicol 1993;72 Suppl 1:4-11

[24] Burnstein MJ

Dietary factors related to colorectal neoplasms.

Surg Clin North Am 1993 Feb;73(1):13-29

[25] Correa P

Human gastric carcinogenesis: a multistep and multifactorial process—First American Cancer Society Award Lecture on Cancer Epidemiology and Prevention.

Cancer Res 1992 Dec 15;52(24):6735-40

6. Lyman, in Primary Care, 1992, in an article on risk factors for cancer commented, "It is no longer reasonable to divide cancers into those that are genetic in origin and those that are environmental in origin. With rare exception, carcinogenesis involves environmental factors that directly or indirectly exert a change in the cell's genome. Virtually all causes of cancer are multifactorial, sometimes involving an inherited predisposition to the carcinogenic effects of environmental factors, which include chemicals, ionizing radiation and oncogenic viruses. Carcinogenesis is a multistep process, including induction, promotion and progression." [26]

7. Jones, et al., in an editorial in the Journal of Cancer Research and Clinical Oncology, 1992, noted that as far as human urinary bladder cancer is concerned, "multifactorial alterations of cellular genes occur." [27]

8. Villa, et al., in the Italian Journal of Gastroenterology, 1991, summarized the multifactorial basis of hepatocellular cancer.[28]

9. Tomatis in IARC Scientific Publications, 1989, presented an overview of perinatal and multigenerational carcinogenesis and commented that, "One of the characteristics of recent decades, which have seen a formidable

---

[26] Lyman GH
Risk factors for cancer.
Prim Care 1992 Sep;19(3):465-79

[27] Jones RF; Debiec-Rychter M; Wang CY
Chemical carcinogenesis of the urinary bladder—a status report.
J Cancer Res Clin Oncol 1992;118(6):411-9

[28] Villa E; Melegari M; Scaglioni PP; Trande P; Cesaro P; Manenti F
Hepatocellular carcinoma: risk factors other than HBV.
Ital J Gastroenterol 1991 Sep-Oct;23(7):457-60

expansion of cancer research, has been the co-existence of the generally agreed hypothesis that most cancers are multifactorial in origin . . . ." [29]

10. Kerenyi, et al., in medical hypothesis postulates that one of the multifactorial bases of the increase in the incidence of cancer is "light pollution." In their opinion, "air pollution, smoking, diet, alcohol, occupational exposures and stress are all considered possible etiologic and risk factors" for cancer. They additionally, suggest that light is a significant factor in carcinogenesis because of changes in melatonin production.[30]

11. Higginson, one of the most significant researchers in the field of respiratory carcinogenesis, in a conference proceedings published in 1988, discusses at greath length the issues of multifactorial carcinogenesis in respiratory and total cancer, and their implication for cancer prevention.[31]

12. On the basis of an overwhelming body of scientific evidence which supports the postion which I have taken, that cancer is a multi-factorial disease, I have outlined the elements of causation which are identifiable from Mr. Joiner's family, personal, medical and occupational his-

---

[29] Tomatis L
Overview of perinatal and multigeneration carcinogenesis.
IARC Sci Publ 1989;(96):1-15

[30] Kerenyi NA; Pandula E; Feuer G
Why the incidence of cancer is increasing: the role of 'light pollution'.
Med Hypotheses 1990 Oct;33(2):75-8

[31] Higginson J
Multifactorial carcinogenesis significance for cancer prevention risk measurement.
Iversen, O. H. (ED.). Theories of Carcinogenesis; International Conference on Theories of Carcinogenesis: Facts, Fashion or Fiction, Oslo, Norway, August 16-20, 1986. New York: Hemisphere Publishing 1988. pp. 322-324.

tory. I have specifically listed these factors in my deposition, I believe that his family history of cancer, his personal history of exposure to cigarette smoke and perhaps to silica, his medical history and his occupational history which included exposure to polychlorinated biphenyls, their contaminants, including polychlorinated bibenzofurans, mineral oil, mineral spirits and other materials together were a sufficient and probable cause of Mr. Joiner's lung cancer. In my opinion, PCBs were a significant contributing cause to Mr. Joiner's lung cancer.

FURTHER THE AFFIANT SAITH NOT.

/s/ Daniel T. Teitelbaum
DANIEL T. TEITELBAUM, M.D.

Sworn to and subscribed before me, on this the 27th day of January, 1993.

/s/ Joan L. Hurley
Notary Public

My commission expires: 11/20/96

Exhibit 1

CURRICULUM VITAE

Daniel T. Teitelbaum, M.D.

Daniel Thau Teitelbaum, M.D., P.C.
155 North Madison
Denver, Colorado 80206
(303) 355-2625

DANIEL THAU TEITELBAUM, M.D.

POSITION:

Clinical Toxicologist, 1988-present
President,
Daniel Thau Teitelbaum, M.D., P.C.
Medical Toxicology & Occupational Medicine

ADDRESS

(BUS): Daniel Thau Teitelbaum, M.D., P.C.
155 North Madison
Denver, Colorado, 80206
(303) 355-2625

(HOME): 2223 S. Alton Way
Denver, Colorado 80231

SCOPE OF PRACTICE:

Clinical Toxicology
Occupational and Environmental Toxicology
Acute and Chronic Poisoning
Analytical Toxicology
Clinical Pharmacology

BORN:

May 26, 1935
New York, New York
Citizenship: U.S.A.

PROFESSIONAL EXPERIENCE:

Qualified Expert Witness in Clinical Toxicology since 1967.

Consultant to the Industrial Commission and State Compensation Fund of Colorado, The United States Food and Drug Administration and the Occupational Safety and Health Administration.

Consultant to industry, agriculture, and labor in occupational and environmental toxicology, including: IBM, CFI Steel, Dresser Industries, W.R. Grace, Coors, Monfort of Colorado. Dynalectron Corporation, Amoco, Xerox, Northern Telecom, Motorola, NCR, TRW, Intel Corporation, Heat and Frost and Asbestos Workers Union, Colorado Construction Trades Council, etc.

Lecturer and seminar leader in all aspects of toxicology practice. Fields of interest: solvents, asbestos, lead, carcinogenesis and biomedical and environmental monitoring.

Extensive experience in the practice of analytical, biomedical and occupational/environmental toxicology. Founder and former director of Poisonlab and Enbionics, independent toxicology laboratories licensed by CDC acredited by AIHA (#60.) Consultant in analytical and clinical toxicology to Bioscience Laboratories and other independent laboratories.

ACADEMIC AFFILIATIONS:

Asociate Clinical Professor of Preventive Medicine, University of Colorado Medical School, Denver, Colorado.

Adjunct Professor of Environmental Sciences, Colorado School of Mines.

Visiting Professor, Medicine and Toxicology, Israel Institute of Technology, The Technion, Haifa Israel.

Scope of teaching: Clinical, occupational and environmental toxicology.

Member of Physicians' Poison Consultation Service	University of Colorado Medical Center, Denver, Colorado

Consultant in Clinical Toxicology — Denver General Hospital and Rockey Mountain Poison Center

CDC licensed Clinical Laboratory Director

1983-1988 Director / Occupational Medicine & Toxicology
Denver Clinic (Accord Medical Center)
701 E. Colfax Ave.
Denver, CO 80203

1982-1983 Staff Physician/Occupational Medicine
Denver Clinic
701 E. Colfax Ave.
Denver, CO 80203

1982-1989 Medical Director, Analytitox Inc.,
Denver, CO

1979-1982 President
Worksafe, Inc.
6825 East Tennessee
Denver, Colorado 80224

1973-1979 President, Poisonlab/Enbionics
Division of Chemed Corporation
1469 South Holly Street
Denver, Colorado 80222

Offices in Denver, San Diego, Cleveland

CLIA #05-1014 AIHA #60

1970-1973 Founder, President, and Toxicology Consultant
Poisonlab, Inc.
1469 South Holly Street
Denver, Colorado 80222

Private Practice—Clinical Toxicology
2045 Franklin Street
Denver, Colorado 80222

1970-1971 Director of Licensed Methadone Treatment Program
IND 6867
2045 Franklin Street
Denver, Colorado 80205

1968-1970 Director of Emergency Services
University of Colorado Medical Center
Denver, Colorado

Assistant Professor of Medicine and Preventive Medicine
University of Colorado Medical Center
Denver, Colorado

1967-1968 Clinical Instructor, Preventive Medicine
University of Colorado Medical Center
Denver, Colorado

EDUCATION:

1956	Bachelor of Arts	Hamilton College Clinton, New York
1960	Master of Hebrew Letters and Rabbi	Jewish Theological Seminary of America
1964	Doctor of Medicine	Albert Einstein College of Medicine
1964-5	Intern, Mixed Medicine	Montefiore Hospital New York City, NY
1965-7	Resident, Internal Medicine	University of Colorado Medical Center, Denver, CO
1967-8	Fellow in Medicine and Toxicology	University of Colorado Medical Center, Denver CO
1991-2	Occupational and Environmental Medicine Program	University of California, San Francisco, CA

COMMITTEES:

Former Member, Toxicology Resource Committee, College of American Pathologists

Former Member, Education Committee, American Academy of Clinical Toxicology

Chairman, United States Food and Drug Administration Advisory Committee on toxicology diagnostic products. (Executive Appointment) 1976-1978

Special Consultant to OSHA, U.S. Department of Labor on Lead, 1977. Participant on behalf of OSHA in lead standards setting hearings

Member, Committee on Operation of Centers, American Association of Poison Control Centers

Chairman, Drug Abuse Committee, American Occupational Medical Association, 1977-1978

Former Member, ASTM Committee E-34 on Safety in the Workplace

Former Member, Board of Trustees, American Academy of Clinical Toxicology

Former Member, Environmental Affairs Committee, W.R. Grace and Co.

Former Member, Chemical Regulations Advisory Committee, Manufacturing Chemists Association

Member, Forensic Sciences Committee of the American Society for Testing and Materials

Member, Occupational Medicine Committee of the American Industrial Hygiene Association

Special Consultant to OSHA, USDOL on Access to Medical Records Standard. 1981

Former Member, State Poison Control Committee, Colorado Department of Health

Former Member, Joint Pesticide Advisory Committee, State of Colorado

Former Secretary-Treasurer to the American Academy of Clinical Toxicology

Former Chairman, Therapeutics Committee, American Academy of Clinical Toxicology

Special Consultant to OSHA, USDOL on Hazard Communication Standard 1982

Special Consultant to OSHA, USDOL on Ethylene Dibromide Standard 1984

Member, Editorial Board, Journal of Toxicology, Clinical Toxicology 1968-1982

Peer Reviewer, Annals of Internal Medicine, 1970-1985

Peer Reviewer, Journal of the American Medical Association, 1975-1985.

Member, Special Blue Ribbon Panel of the Executive Office of the President, National Science Foundation/Council on Environmental Quality on Future Health Implications of Emerging Technologies, 1984

Secretary of the Medical Executive Committee, St. Joseph Hospital, Denver, Colorado 1985-1986

Special consultant, USDOL, OSHA, on Benzene Standard, 1986

SOCIETIES:

American Academy of Clinical Toxicology
American Association of Poison Control Centers
American Academy of Forensic Sciences
The American College of Clinical Pharmacology
American Industrial Hygiene Association
American Society for Testing and Materials
American Academy of Veterinary Toxicologists
American Society of Clinical Pathologists

Forensic Science Society
Rocky Mountain Academy of Industrial Medicine
Occupational Medical Association
Society of Sigma Xi, The Scientific Research Society
Denver Medical Society
Colorado Medical Society
American Association for the Advancement of Science
Society for Risk Analysis

HONORS:

World Health Organization Traveling Fellowship in Clinical Toxicology
Founders Award, American Academy of Clinical Toxicology
Student Fellowship, Jackson Memorial Laboratory, 1952/3
Numerous Academic Prizes in College

BOARD CERTIFICATION:

Board Certified American Board of Medical Toxicology

Recertified by examination, August, 1976

FELLOWSHIPS:

Fellow, American College of Clinical Pharmacology, 1973

Fellow, American Academy of Clinical Toxicology, 1976

PUBLICATIONS:

USE OF SYRUP OF IPECAC, Rocky Mountain Medical Journal, June, 1967.

DETAILING IPECAC, Journal of Pediatrics, November, 1967.

POISONING DEATHS TOO HIGH IN U.S. (corr.) NEJM, November 9, 1967.

STRAMONIUM POISONING IN "TEENY-BOPPERS", Annals of Internal Medicine, January, 1968.

NEW DIRECTIONS IN POISON CONTROL, Clinical Toxicology, 1(1):3-13, 1968.

MANAGEMENT OF SNAKE BITE INJURIES, Rocky Mountain Medical Journal, October, 1968.

ARSINE POISONING, Archives of Environmental Health, July, 1969.

ELEMENTAL MERCURY SELF POISONING, Clinical Toxicology. September 1969 with Dr. John E. Ott.

VEGETABLE OIL AEROSOL SPRAY POISONING, Rocky Mountain Medical Journal, October, 1969.

MISDIAGNOSIS OF DRUG ABUSE (lett.), JAMA, December 16, 1969.

LONG HAIR, PECULIAR BEHAVIOR (Corr.), Lancet, November 18, 1969.

ACUTE STRYCHNINE INTOXICATION, Clinical Toxicology, June, 1970 with Dr. John E. Ott.

ISONIAZID SELF POISONING, Neurology, April, 1970 with Dr. David Terman.

POISONING WITH PSYCHOACTIVE DRUGS, Ped. Clin. N.A., August, 1970.

MANAGEMENT OF POISONING WITH ANTIDEPRESSANT DRUGS, Clinical Toxicology, September, 1971 with T. Slovis, J. Ott, et al.

POISONING—Chapter in Current Pediatric Diagnosis with Dr. John Ott, 1970 and 1972 editions.

METHADONE MAINTENANCE IN A PRIVATE PRACTICE SETTING—American Academy of Clinical Toxicology, August, 1972, Clinical Toxicology 6:2 (1973).

MANAGEMENT OF ORGANOPHOSPHORUS PESTICIDE POISONING. New York State Society of Anesthesiologists, December, 1972.

ROUND THE CLOCK SCREENING FOR DRUGS USING THE EMIT SYSTEM, Daniel T. Teitelbaum, Gyorgy Vidacs, Clinical Toxicology 1973.

TOXICOLOGY MEDICAL AND LABORATORY SERVICES IN THE INDUSTRIAL SETTING: in Guidelines for Analytical Toxicology Programs. vol. I., edited by J.J. Thoma, et al. CRC Press, Cleveland, 1977.

DIAGNOSIS AND MANAGEMENT OF RECREATIONAL, MESCALINE SELF POISONING, D. T. Teitelbaum, M.D. and D. C. Wingeleth, Ph.D., Journal of Analytical Toxicology, January/February, 1977.

NONCONCORDANCE BETWEEN CLINICAL IMPRESSION AND LABORATORY FINDINGS IN ACUTE POISONING, with Janet Morgan, M.A., and Gail Gray, M.A., Clinical Toxicology. No. 4, 1977.

ACETAMINOPHEN: CLINICO-PATHOLOGIC CORRELATIONS, A.A.C.C., Nov. 1979.

Contributor "The College of American Pathologists Toxicology Program" by David Sohn. Journal of Analytical Toxicology. Vol. 1, May/June 1977, p. 111-117.

REASSURANCE PRESCRIBED FOR NONTOXIC INGESTIONS, St. Anthony Hospital Medical Staff News, Denver, Colorado, July/August, 1979.

INSULATE YOUR HOME WITH CAUTION, with Sheila Teitelbaum, M.A., National Health, September, 1979.

HOW ADEQUATE IS ADEQUATE VENTILATION?, with Sheila Teitelbaum, M.A., National Health, October, 1979.

INFORMATION AND THE PREVENTION OF GENETIC INJURY, Proceedings, National Safety Congress, 1979.

MICROWAVE OVENS: KEEPING THE RISKS LOW, with Sheila Teitelbaum, M.A., National Health, November, 1979.

CLINICAL RECOGNITION OF ASBESTOSIS, in Occupational Diseases: Steps for Positive Action, Hour 2, Home Continuing Medical Education, 15 Columbus Circle, NY 10023.

WORKING WITH LEAD, with Ralene Reynolds, M.A., The Center for Toxicology, Man and Environment, Inc., May, 1980.

THE TOXICOLOGY OF MASS CHEMICAL ACCIDENTS, proceedings of the First International Symposium of the Israel Society of Critical Care Medicine, Haifa, June, 1980.

ACETAMINOPHEN POISONING IN CONTEMPORARY PRACTICE, California, F.P., June, 1982.

HOBBIES AND HAZARDS, with Sheila Teitelbuam, Consultant, March 1982.

TRW HEALTH AND SAFETY MANUAL, with Ralene Reynolds and Sheila Teitelbaum, in cooperation with TRW Semiconductor Corporation, Lawndale, California, September, 1981.

SEMICONDUCTOR PRODUCTION CAN BE NASTIER THAN YOU THINK, with Sheila Teitelbaum, Consultant, September, 1982.

ASPIRIN AND ACETAMINOPHEN POISONING: DIAGNOSIS AND CLINICAL MANAGEMENT, with Sheila Teitelbaum, Consultant, November, 1982.

ROLE OF THE PHYSICIAN IN THE DEVELOPMENT OF TOXIC SUBSTANCES HAZARDS COMMUNICATION PROGRAMS, 2nd World Meeting of Clinical Toxicology, Vet Hum Toxicology 24(4):279, 1982.

TOXICOLOGIC IMPLICATIONS OF GROUNDWATER CONTAMINATION, Motorola Corporation, 1983, company confidential.

EVALUATION OF BENEFITS OF DRUG ANALYSIS IN THE ROUTINE CLINICAL MANAGEMENT OF ACUTE POISONING, J Toxicol Clin Toxicol 22(6):589-92, 1984.

HUMAN HEALTH IMPLICATIONS OF EMERGING CHEMICAL TECHNOLOGIES, Proceedings of a National Science Foundation/Council on Environmental Quality special commission, September, 1984, Preliminary Publication March, 1985 (NSF/CEQ).

LABORATORY MANAGEMENT OF ACUTE POISONING, Diagnostic Medicine, in publication, Scheduled for late 1985.

ALCOHOL AND CRASHES, American Bar Association, 1st Annual Seminar on Transportation Negligence, Seminar Handbook, ABA 1985.

PHOTOACTIVE CHEMICALS USED IN PHOTORESIST SYSTEMS. State Art Rev Occup Med 1(1):59-68, 1986.

LEAD POISONING IN A CAPACITOR AND RESISTOR PLANT-COLORADO, MMWR 1985; 34(25):384-85. with Alkes I; Kadushin F; Lampert K; Hopkins R; Ryan J; Novotny T.

NEUROBEHAVIORAL FINDINGS IN TWO CASES OF CHRONIC LOW LEVEL CARBON MONOXIDE POISONING, Vet Hum Toxicol 29(6):479, 1987, with AC Bronstein, FS Kadushin.

NEUROPSYCHOLOGICAL DYSFUNCTION FROM ACUTE AND LONG TERM ORGANIC CHEMICAL EXPOSURES: REPORT OF SIX CASES, Vet Hum Toxicol 29(6):477, 1987, with FS Kadushin, AC Bronstein, DM Baker.

DETECTION OF UNSUSPECTED, NON-FAVA BEAN SENSITIVE G6PD DEFICIENCY IN SEMICONDUCTOR FABRICATION WORKERS IN ISRAEL. Proceedings WHO conference, "Consultation on genetic predisposition to toxic effects of chemicals", Krefeld, FRG, 17-20 October 1989. with B. Raikhlin.

ASBESTOSIS IN RETIRED STEAM RAILWAY CRAFTSMEN. Documentation of Exposure to Asbestos from Photographs in the Library of Congress Prints and Photographs Collection. Bulletin de la Societe des Sciences Medicales du Grand-Duche de Luxembourg. Proceedings of the International Congress on Clinical Toxicology, Poison Control and Analytical Toxicology LUX TOX '90, 2-5 May 1990, Luxembourg. pp. 325-330.

AMBIENT AIR POLLUTION FROM INDUSTRIAL SOURCES. in Air Pollution in Central and Eastern Europe: Health and Public Policy. BS Levy, ed. Management Sciences for Health, Boston, MA: 1991. p.36

REGULATION: LAWS AND GUIDELINES. in Air Pollution in Central and Eastern Europe:

Health and Public Policy. BS Levy, ed. Management Sciences for Health, Boston, MA: 1991. p.145

TEACHING VIDEOTAPES:

"Chemical Safety", produced by Hewlett Packard.
"Primary Management of the Acutely Self-Poisoned Adult", produced by the Network for Continuing Medical Education.

"Occupational Diseases: Steps for Positive Action", produced by the Network for Continuing Medical Education.

"Management of the Acutely Poisoned Patient", produced by BioScience Laboratories, Van Nuys, California.

"The Six Big Ones", in cooperation with Xerox Microelectronics Center, November, 1981.

A Needle in a Haystack. The role of the laboratory in the management of the poisoned patient. Bio-Science Laboratories, 1982.

"Definitive Management of Poisoning", BioScience Laboratories, Van Nuys, California, October, 1982.

"ZAP: Human Electrocution—What Happens and How to Prevent It", in cooperation with Motorola BiPolar Division, Mesa, Arizona 1982.

"Diagnosis and Management of Substance Abuse Disorders." NCME, NY, 1983.

PUBLISHED BIBLIOGRAPHIES:

With B. J. Croall, M.A.

THE HEMATOLOGIC EFFECTS OF TRIAZINES, October, 1978.

BENZIDINE AND HAIR DYES, April, 1978.

HEMATOLOGIC EFFECTS OF EXPOSURE TO PESTICIDES, February, 1978.

TOXICITY OF CYCLONITE, August, 1978.

ALPHA METHYLDOPA, February, 1978.

SUGESTED TITLES FOR AN INDUSTRIAL TOXICOLOGY LIBRARY, June, 1977.

PAINT REMOVERS AND CARBON MONOXIDE INTOXICATION, April, 1977.

EXPOSURE TO SULFURIC ACID, October, 1977.

CONTACT DERMATITIS, December, 1977.

ANTICONVULSANTS, January, 1976.

ANALYTICAL ASPECTS OF LEAD INTOXICATIONS, October, 1976.

STANDARD INTERNATIONAL (SI) UNITS, June, 1979.

PENTAZOCINE ADDICTION, April, 1981.

TOXICITY OF THALLIUM, June, 1982.

OCCUPATIONAL ASTHMA, September, 1982.

IN THE UNITED STATES DISTRICT COURT
FOR THE NORTHERN DISTRICT OF GEORGIA
ATLANTA DIVISION

(Title Omitted in Printing)

PLAINTIFFS' RESPONSE TO DEFENDANTS' STATEMENT OF MATERIAL FACTS AND STATEMENT OF MATERIAL FACTS WHICH REMAIN IN ISSUE

DEFENDANTS' FACTS AND PLAINTIFFS' RESPONSE

1.

Robert K. Joiner has worked at The City of Thomasville Water & Light Department ("Thomasville Water & Light") since 1973.

Plaintiffs admit this fact and further show the Court that this has been Robert Joiner's only employment since his graduation from high school. During this employment he was continually and regularly exposed to electrical transformers which were contaminated with dangerous and carcinogenic levels of polychlorinated biphenyls ("PCBs"), polychlorinated dibenzofurans ("dibenzofurans" also sometimes referred to as "PCDF"), and polychlorinated dibenzo-p-dioxins ("dioxins" also sometimes referred to as "PCDD").

2.

As part of his employment at Thomasville Water & Light, Mr. Joiner worked with and around electrical transformers and voltage regulators.

Plaintiffs admit this fact and further show the Court that working on electrical transformers took up over 50% of Robert Joiner's work time. Over 50% of the transformers which have been tested since 1986 have had

levels of PCB in the dielectric fluid to which Mr. Joiner was regularly exposed.

3.

The dielectric fluid contained in the electrical transformers and voltage regulators used by Thomasville Water & light was mineral oil.

Plaintiffs deny that this fact is true and further show the Court that while the dielectric fluid was *supposed* to be mineral oil, more than 50% of the transformers contained levels of PCB contamination which were unsafe and which contributed to the cause of Robert Joiner's lung cancer. This percentage is based only on transformers tested after 1986. Mr. Joiner began working with contaminated transformers in 1973 and it is probable that an even higher percentage were contaminated in the early years of his exposure.

4.

After Thomasville Water & Light began testing its mineral oil-filled equipment for the presence of PCB contamination in 1983, it determined that less than twenty percent of its equipment was contaminated with PCBs.

Plaintiffs deny that this fact is true and further show the Court that more than 50% of the transformers contained levels of PCB contamination which were unsafe and which contributed to the cause of Robert Joiner's lung cancer. The term "contaminated" is a regulatory term as used by Defendants and does not include the numerous transformers which contained PCB but did not qualify as PCB "contaminated" under regulations which govern industry and which are, by their very nature, compromises.

5.

Of all of the equipment tested by Thomasville Water & Light, only about 2.5% of the transformers tested contained levels above 500 ppm, while only about 16.7% of the equipment contained levels above 50 ppm.

Plaintiffs admit that this fact is true for the statistics provided but are unable to admit that it is representative of the percentage of transformers which contained PCB in the earlier years of Mr. Joiner's employment and believe that the percentage would have been higher then. Plaintiffs further show the Court that while the dielectric fluid was supposed to be mineral oil, more than 50% of the transformers contained levels of PCB contamination which were unsafe and which contributed to the cause of Robert Joiner's lung cancer.

6.

The EPA considers any electrical transformer containing less than 50 ppm of PCBs to be a "Non-PCB Transformer" and does not regulate such equipment.

Plaintiffs admit that this fact is true and further show the Court that regulations which govern industry are, by their very nature, compromises and do not relieve Defendants from their duty to prevent the contamination in the first place and then to warn of the contamination once it was known to them. Further, such regulations are irrelevant to measuring Defendants' conduct or duty or liability because Defendants have admitted that there should have been 0 ppm in the supposedly pure mineral oil dielectric. Defendants were well aware of PCB contamination of mineral oil dielectric fluids before Robert Joiner graduated from high school in 1973 and before he was ever exposed to the PCB which caused his injuries.

7.

One (1) transformer at Thomasville Water & Light (not manufactured by either GE or Westinghouse) contained 1,880 ppm PCBs, which was the highest reported level for any transformer or voltage regulator.

Plaintiffs admit that the records which are available show this to be true and further show that regardless of

whether this transformer was manufactured by Defendants GE or Westinghouse, the PCB was certainly manufactured by Defendant Monsanto, the only manufacturer of PCB in the United States. Plaintiffs further show that had Defendants GE and Westinghouse warned of the existence of PCB in their mineral oil dielectric that Plaintiff would have exercised appropriate cautions even when handling the transformers manufactured by other, smaller, less sophisticated manufacturers.

8.

In 1991, Robert K. Joiner was diagnosed with small cell lung cancer.

Plaintiffs admits that Mr. Joiner has cancer of the lung and that he was only 37 years old when diagnosed. However, a description of the particular cancer as being "small cell" cannot be admitted as lung cancer cannot readily be catalogued this simply.

9.

Mr. Joiner smoked cigarettes, and both of his parents smoked cigarettes while he was growing up in their home; thus, according to the plaintiffs' experts, Mr. Joiner had consistent, substantial exposure to cigarette smoke for many years.

Plaintiffs admit that Robert Joiner smoked cigarettes and that his parents smoked cigarettes. Plaintiffs deny that this can be, or has been, characterized as a substantial exposure to cigarette smoke. Plaintiffs further show this Court that Plaintiffs' experts have opined, to a reasonable degree of medical certainty, that cigarettes were merely the initiators of the cancer which is killing Robert Joiner. PCB exposure exponentially increased Robert Joiner's risks of cancer.

10.

Small cell cancer of the lung in humans is caused by tobacco smoke.

Plaintiffs deny this fact to the extent that Defendants seek to imply that tobacco smoke is the only source of the kind of cancer suffered by Robert Joiner. In fact, the kind of cancer suffered by Robert Joiner is associated with various things including exposure to chemicals. Robert Joiner's lung cancer was caused by exposure to PCBs.

11.

According to the plaintiffs' own experts, there is no credible evidence as a scientific probability that PCBs cause or promote small cell lung cancer in humans.

Plaintiffs deny that this fact is true. In fact, the experts retained by Plaintiffs have opined that Robert Joiner's lung cancer was promoted, and thus caused, to a reasonable degree of scientific and medical certainty, by his exposure to PCBs. Further, Plaintiffs' experts have identified to the Court studies which support their opinions as well as reasons, which are beyond the studies, supporting the proposition that PCB promotes and thus causes lung cancer.

12.

According to the plaintiffs' own experts, there are no epidemiological studies which conclude that PCBs cause or promote small cell lung cancer in humans, or, more generally, any form of lung cancer in humans.

Plaintiffs deny that this fact is true. In fact, at least the following studies are instructive on this point and materially contradict Defendants' arguments and support Plaintiffs' position that a genuine issue of fact remains in this case.

Cancer Mortality of Capacitor Manufacturing Workers, Bertazzi et. al., American Journal of Industrial Medicine 11:165-176 (1987)

> "The multiform toxic action of PCBs has been documented in numerous experimental studies, without however, identifying a specific mechanism." 166

> 'Studies of the metabolic fate of these substances in animals sustain the plausibility of a carcinogenic action." 166

> "a previous study [Puccinelli, 1954] showed that less than six months employment could result in substantial exposure to PCBs." 168

> "deaths owing to cancer were significantly higher than expected according to either the national or the local population mortality rates." 169

> "Even the three cases of death from lung cancer were more than expected, but not significantly so." 169

"Table IV. Mortality From Selected Causes of Male Workers Exposed to PCBs"

Cause of death (ICD 8th revision)		National		Local	
	Observed	Expected	SMR	Expected	SMR
Lung Cancer p.170	3	1.2	250	1.6	187"

> **"Analysis by duration of exposure, latency, and year of first exposure did not reveal any definite pattern or trend of mortality for any of the relevant causes." 170**

"Cancer type/ site	*Age at hire(y)*	*Year of hire*	*Length of exposure (y)*	*Latency (y)*	*Age at death (y[)]*
Lung (162)	60	1951	6.7	26	86
Lung (162)	28	1954	.1	7	35
Lung (162) p.171	38	1962	.5	19	57"

Mortality of PCB Workers at the Monsanto Plant in Sauget, Illinois, Zack and Musch, 12/14/79.

> "The SMR for lung cancer was high at 278." JOI 003188

> "There were three deaths observed from lung cancer with 0.94 expected, yielding a SMR of 319." JOI 003188

Table 4

Observed and Expected Deaths. All Males

I.C.D. No. (Eighth Rev.)	Cause of Death	Observed	Expected	SMR
162, 163	Lung	4	1.44	278

Number of persons observed = 89

Table 5

Observed and Expected Deaths, White Males

I.C.D. No. (Eighth Rev.)	Cause of Death	Observed	Expected	SMR
162, 163	Lung	3	0.89	337

Number of persons observed = 60

Table 6

Observed and Expected Deaths, Nonwhite Males

I.C.D. No. (Eighth Rev.)	Cause of Death	Observed	Expected	SMR
162, 163	Lung	1	0.55	182

Number of persons observed = 29

Table 7 shows that all victims of lung cancer had exposures of more than 2 years and four months and less than 3 years.

The Eppley Institute for Research in Cancer was asked by Monsanto to review statistics relating to its workers exposed to PCBs. The results are documented by Dr. E. Mahboubi, MPH, Professor and Head of Epidemiology, at the Institute in letters to Dr. F. R. Johannsen, chief toxicologist for Monsanto:

> Letter of Ocober 17, 1975, page 2:
>
> [T]he lung cancer mortality for these employees, particularly for birth cohorts of 1890-1909, appears to be in excess of what one may expect it to be.
>
> I, as a physician, duly understand your concern for maintaining the confidentiality of these records and I have not duplicated the material. If you wish to have this material returned to you, please so indicate.

> Letter of November 19, 1975, page 1:
>
> The mortality due to lung cancer, however, ranges from betwen three- and ten-fold, when compared to the figure expected, and the increased rate is significant.
>
> Furthermore, all six of the lung cancer victims worked for less than 3 years in the K[rummrich] plant.

In an internal memorandum, Dr. Johannsen had the following to say about PCBs and lung cancer in Monsanto's PCB workers to George Roush, an employee of GE:

Memorandum

From: Dept. of Medicine & Environmental Health—F.R. Johannsen

Date: August 27, 1976 (note this is after the report of Dr. Mahboubi which, though it found lung cancer at high rates, did not think the lung cancer rate was significant.)

Subject: Epidemiology

Reference: Krummrich Plant

In a study of 140 PCB exposed employees, 23 died of all causes and "deaths attributed to lung cancer—4".

> When compared to the 1969 U.S. male population, this group of 23 deaths would be expected to contain 1.24 cases attributable to lung cancer. Statistical comparison of the observed lung cancer versus the expected lung cancer yielded a highly significant cni X^2 value of 7.1. This number is even more significant that the value (cni X^2- 6.8) obtained following evaluation of the full 50-person mortality population.

This gives an SMR of 322!

Ikeda, et. al., *A Cohort Study on Mortality of Yusho Patients—A Preliminary Report*, Fukuoko Acta Med., 78: 297-300, (1987) is a study of Yusho victims exposed to PCBs, as summarized by the World Health Organization's International Programme on Chemical Safety, *Environmental Health Criteria 140, Polychlorinated Biphenyls and Terphenyls (Second Edition)* at page 499, has the following finding:

> "A statistically significant excess mortality was seen for malignant neoplasms, cancer of the liver and cancer of the lung, trachea, and bronchi in males . . . ."

13.

According to a laboratory test of Mr. Joiner's adipose (fat) tissue obtained by Mr. Joiner in 1991 approximately four months after the diagnosis of his small cell lung cancer, Mr. Joiner's body level of PCBs was .3 ppm.

Plaintiffs admit that this is the finding of the laboratory and further show that this finding absolutely shows exposure to PCBs. More significantly, this finding is not believed by any credible scientist as having any meaning or significance as the laboratory work is of questionable quality, (it found no detect levels of dioxins even though dioxins are almost certainly in the tissues of every human in North America), were not properly standardized to World Health Organization or Centers for Disease Control criteria, were taken after radiation, chemotherapy, and rapid weight loss and thus there is no control group to which the measurements have been, or can be, compared. Levels of PCBs, like any other level, are only relevant when compared to norms or expected standards. There are no such standards which include anyone with Mr. Joiner's medical history. Finally, as years had passed since Mr. Joiner's last exposure, it is quite possible that his

levels simply decreased with time—this is a well documented phenomenon in PCB exposure victims.

14.

Mr. Joiner's body level of PCBs, as measured in the analytical laboratory test of Mr. Joiner's adipose tissue, shows that Mr. Joiner's body level of PCBs was slightly less than the average individual in North America who has not had any occupational exposure to PCBs.

Plaintiffs deny that these laboratory results can be used for any valid scientific extrapolation except to show that, assuming that the test was conducted properly, Mr. Joiner positively has PCB in his tissues. PCB does not exist in nature. Only Monsanto manufactured PCB in the United States. Thus, any PCB in Robert Joiner is there because of the conduct of Defendant Monsanto if not all three Defendants.

15.

None of the plaintiffs' expert witnesses can quantify either Mr. Joiner's exposure to PCBs or his dose of PCBs.

Plaintiff deny that this fact is relevant. It is impossible to exactly quantify Mr. Joiner's exposure to PCBs or his dose of PCBs except to note that 50% of his time was spent working on transformers which had been exposed to high heat occurrences including fires. It is not necessary for experts to be able to so quantify exposure so long as exposure occurred—which Defendants implicitly admit must have happened. Further, more than 50% of these transformers were contaminated with some level of PCBs. Given the fact that PCBs are carcinogens, there is no safe level to which Mr. Joiner could be exposed. As an example of the extent of exposure, if a transformer was contaminated with PCBs at only one part per quadrillion (10^{15}) instead of the fifty parts per million (10^{6})

to which he was actually exposed on a regular basis, Mr. Joiner, in a full breath of air in which he would inhale about ten billion trillion molecules, would inhale ten million PCB molecules. (Brown, Michael H., *The Toxic Cloud*, 249-250 (1987))

FACTS WHICH REMAIN IN ISSUE

In addition to the facts set forth above, which remain hotly contested, the following material facts remain at issue and must be resolved by a jury of Plaintiffs' peers.

1.

Whether Defendant Monsanto manufactured all of the PCB to which Robert Joiner was exposed remains in issue.

2.

That Defendants GE and Westinghouse manufactured transformers which contained PCBs to which Robert Joiner was exposed remains in issue.

3.

That the PCB to which Robert Joiner was exposed was contaminated with polychlorinated dibenzofurans, and polychlorinated dibenzo-p-dioxins remains in issue.

4.

That Defendants were aware of the dangers of PCBs and PCDFs and PCDD when Robert Joiner was exposed to these chemicals remains at issue. PCDF and PCDD are expected to be formed when PCB is subjected to high heat and fire remains in issue.

5.

Given Robert Joiner's history of exposure to cigarette smoke he had a very minimal chance of lung cancer at

37 years of age related solely to tobacco smoke exposure remains in issue.

6.

That Robert Joiner suffered lung cancer at 37 years of age because of the known promotional effects of PCBs and PCDFs to which he was exposed remains in issue. That it is more likely than not, to a reasonable degree of medical and scientific certainty, that Robert Joiner's lung cancer was caused by the conduct and chemicals manufactured by Defendants remains in issue.

7.

The credibility of Plaintiffs' experts opinions, and the credibility of the conclusions drawn by them, remain in issue for determination by the jury.

8.

The credibility and weight to be given the studies which demonstrate a positive correlation between exposure to PCBs and lung cancer remains a question for determination by the jury.

9.

The meaning of the Triangle Laboratories adipose tissue results remains a question of weight and credibility to be determined by the jury. (Assuming, of course that Defendants can make a showing that the results are admissible. In all likelihood this showing can not be made because the methodology is seriously flawed.)

10.

That small cell cancer of the lung is humans is a signature disease of exposure to tobacco smoke is a fact remaining in issue.

11.

Mr. Joiner's smoking history is also a fact which remains in issue.

12.

Whether or not valid epidemiological studies exist which demonstrate a positive correlation between exposure to PCBs and lung cancer remains in issue.

13.

Whether or not animal studies can be reasonably relied upon to be indicators of human health efffects remains in issue.

BURGE & WETTERMARK

/s/ Michael J. Warshauer
MICHAEL J. WARSHAUER
Georgia State Bar No. 018720

2700 The Grand
75 Fourteenth Street, Northeast
Atlanta, Georgia 30309
(404) 875-2500

IN THE UNITED STATES DISTRICT COURT
FOR THE NORTHERN DISTRICT OF GEORGIA
ATLANTA DIVISION

(Title Omitted in Printing)

AFFIDAVIT OF JOANNE BEAUVOIR BROWN

Personally appeared before me, the undersigned officer duly authorized to administer oaths, Joanne Beauvoir Brown, who after being duly sworn, deposes as follows:

1.

My name is Joanne Beauvoir Brown and I am of the age of majority and am competent to give this affidavit.

2.

The facts stated in this affidavit are based upon my own personal knowledge.

3.

I am an attorney with the law firm of Freeman & Hawkins and along with Joe C. Freeman, Jr., represent Monsanto Company in this matter.

4.

In July, 1993, our office provided to Dr. Anna-Luise A. Katzenstein, M.D. of the State University of New York Health Science Center in Syracuse, tissue slides from the left scalene node biopsy of the Plaintiff in this case, Robert K. Joiner, for her review.

5.

Based upon her review of the stained slides, she was able to confirm the nature of the tumor and support the diagnosis of small-cell carcinoma.

6.

She summarized her diagnosis and findings in her pathology consultation report of which we were provided a copy.

7.

On August 27, 1993, a "meet and confer" was scheduled at the offices of Burge & Wettermark between counsel for Plaintiffs and counsel for Monsanto to address and attempt to resolve disputes which had arisen with respect to certain of Plaintiffs' discovery requests.

8.

In attendance at that meeting were Joe C. Freeman, Jr., and I representing Monsanto and Michael Warshauer and James Holland, II, representing the Plaintiffs.

9.

During the "meet and confer", Joe Freeman and I advised Mr. Warshauer that we had consulted with a pathologist in Syracuse, New York, who had reviewed the tissue slides from Robert Joiner to confirm the diagnosis of small-cell lung cancer.

10.

Joe Freeman told Mr. Warshauer that we had a report from the pathologist confirming that Mr. Joiner had small-cell lung cancer. A true and correct copy of the pathology report is attached as Exhibit "A".

Further, affiant sayeth not.

/s/ Joanne Beauvoir Brown
JOANNE BEAUVOIR BROWN

Sworn to before me this 25th day of February, 1994.

/s/ Diane M. Casey
Notary Public, Fulton County, Georgia
My Commission Expires November 15, 1997

08/03/93
10:26

SURGICAL PATHOLOGY CONSULTATION,
(315) 464-4750
SUNY HEALTH SCIENCE CENTER
DEPT of PATHOLOGY, F.R. DAVEY, MD,
CHAIRMAN
750 EAST ADAMS STREET, SYRACUSE, NY 13210

NAME: JOINER, ROBERT K
PAT#: APC-11949
ACCT: 0000000

PATHOLOGY NO.: S93-05814
PATIENT LOCATION: PVT OUT
ATTENDING PHYSICIAN:
ORDERING PHYSICIAN:

DATE OF SURGERY: NOT GIVEN
DATE RECEIVED: 07/22/93 AGE/SEX: 37Y M
REPORT DATE: 08/03/93 DOB: 01/01/1956

SPECIMEN:

CLINICAL HISTORY: This 37 year old man presented with a mediastinal and left lung mass. A left scalene lymph node biopsy was performed.

DIAGNOSIS: Soft tissue, left scalene area, biopsy—

METASTATIC SMALL CELL CARCINOMA
(see microscopic description)

AAK/Cad

T-1X000 M-80416 CODE: I

GROSS DESCRIPTION:

Four unstained slides (91S-6861) are received from John D. Archbold Memorial Hospital, Thomasville, GA.
AAK/cad

MICROSCOPIC DESCRIPTION:

We performed routine H&E staining on one slide and immunohistochemical staining for cytokeratin, T-cell antigen (UCHL) and B-cell antigen (L26) on the others. The routine H&E stained slide shows a poorly differentiated malignant tumor present within soft tissue. There is extensive crush artifact that makes interpretation difficult. However, the tumor is composed of small cells with dispersed nuclear chromatin pattern and scant cytoplasm. The cells infiltrate between adipose tissue and lack cohesion. The differential diagnosis on the routine stain is between a small cell carcinoma and a lymphoblastic lymphoma. Our immunohistochemical stains show weak staining in the cells for cytokeratin and negative staining for the lymphoid markers. These findings confirm the epithelial nature of the tumor and support a diagnosis of small cell carcinoma.

/s/ Anna-Luise A. Katzenstein, M.D.
ANNA-LUISE A. KATZENSTEIN, M.D.
Attending Pathologist

Also Seen By:
Copy To: Not applicable

MEDICAL RECORD COPY—DO NOT REMOVE

IMPORTANCE OF 10C AND
10CA INSULATING OIL

General Electric's 10C and 10CA oil helps dissipate the heat in induction apparatus; and aids in rupturing the arc in oil circuit breakers. In both cases, 10C and 10CA oil is a major part of the insulation of the apparatus, and this oil is compatible with all materials in the apparatus. (Only 10C and 10CA oil should be used in General Electric apparatus.) Over 60 years of careful observation, research, and development of insulating oils have brought about the high quality and long-life of GE 10C and 10CA oils. The longer an oil can maintain good physical, chemical, and electrical qualities in an apparatus, the less

the maintenance costs will be for testing the oil. Where quality and durability are concerned, GE 10C and 10CA oil have no equal.

Progressive improvements have been made in the oil over the years. One recent change during 1961 was an increase in flash point of 10C and 10CA oil from 130° minimum to 145C minimum, to provide a greater margin of safety in thermally-uprated transformers.

* * * *

CHARACTERISTICS AND SCREENING SPECIFICATIONS

The following physical, chemical, and electrical properties of 10C and 10CA oils as shipped are controlled to maintain continuity and quality indicated by these tests. Methods for these tests are included in ASTM D117.

Dielectric Strength	Min. 30 kv	Average 35 kv
Flash Point	Min. 145C	Average 150C
Pour Point	Below –40C	Average –54C
Viscosity at 37.8C (Saybolt)	Max. 62 sec	Average 59 sec
Viscosity at OC	Max. 320 sec	Average 290 sec
Specific Gravity. 15.5/15.5C	0.865 to 0.905	Average 0.890
Total Acid Number	Max. 0.02	Average less than 0.01
Color	Max. 0.5	Average less than 0.5
Power Factor. 60 cycle @ 100C	Max. 0.30%	Average 0.1%
Water Content	Max. 30 ppm	Average 20 ppm
Free Chloride Ion	Max. 0.10 ppm	Average none
Free Sulfate Ion	Max. none	Average none
Interfacial Tension, 25C	Min. 40	Average 46
Total Combined Sulfur	Max. 0.15	Average 0.06
Corrosive Sulfur Compounds	Max. non-corrosive	Average non-corrosive
Visual Condition	Clear	Average clear
DBPC Inhibitor Content (for 10CA only)	Min. 0.15	Average 0.17

Note: Additional processing is required for oil to be used in certain high voltage apparatus. These special requirements are covered in such bulletins as GEI 28004, GEI 65070, and GEI 70353.

Charles I. Hubert
professor of electrical engineering
united states merchant marine academy

preventive maintenance of electrical equipment

second edition

McGraw-Hill Book Company
New York St. Louis San Francisco London
Sydney Toronto Mexico Panama

* * * *

2-20 Drying Electrical Insulation

The most favored method of drying insulation is through the application of external heat. This is conveniently done with a regular baking oven, as shown in Fig. 2-18, or by means of an improvised oven of tarpaulin surrounding the machine. A vent at the top of the tarpaulin permits the exit of moisture-laden air. Small machines are often successfully dried by placing them on top of a boiler. When external heat is used for baking, the temperature of the windings should not exceed 90°C as measured by thermometers taped to the coils. The best source of heat for makeshift ovens is an electric heater or radiant-heat lamps; steam heaters or hot-air furnaces also do an adequate job.

Sixty-second insulation-resistance measurements should be recorded every 4 hr during the drying-out process. A typical drying curve for a dc motor armature is shown in Fig. 2-19. During the first part of the drying operation the increase in temperature causes a decrease in the indicated values of insulation resistance. Then, with a constant drying temperature, the resistance increases as the moisture is driven out. When the insulation is dried and allowed to cool, the resistance increases to a high value. The plotted values of insulation resistance are not corrected for temperature, because such correction would serve no useful purpose in this case.

* * * *

INSTRUCTIONS

GEI-32190F

SECONDARY UNIT

SUBSTATION TRANSFORMERS

Oil-Immersed

* * * *

DRYING A TRANSFORMER

After drying the oil in a filter press, the moisture remaining in the windings and insulation can be driven off by heating the transformer. This can be accomplished by shorting one winding and applying a suitable voltage on the other. Full-load current can be obtained by applying the impedance volts of the transformer. *Be sure to load the entire winding.* If the transformer is at room temperature at the start of drying, 125 percent load may be applied until the top oil temperature reaches 65 C. At this point, the current should be reduced in accordance with the following table:

Maximum Allowable Short-circuit Amperes in Percent of Full Load	Maximum Allowable Top Oil Temperature in Degrees C
100	75
85	80
50	85

Since the windings are at a higher temperature than the oil, the insulation may be damaged if these values are exceeded. Filtration during the heat run will not greatly hasten the drying process because at these temperatures the filter press loses its ability to remove any appreciable amount of moisture.

* * * *

FOOD AND DRUG ADMINISTRATION'S REGULATIONS FOR PCBs IN VARIOUS COMMODITIES

Commodity	Allowable Levels (ppm)
Milk	1.5
Manufactured dairy products	1.5
Poultry	3.0
Eggs	0.3
Finished animal feeds	0.2
Animal feed components	2.0
Edible portion of fish/shellfish	2.0
Infant and junior foods	0.2
Paper and packaging material	10.0

IN THE UNITED STATES DISTRICT COURT
FOR THE NORTHERN DISTRICT OF GEORGIA
ATLANTA DIVISION

(Title Omitted in Printing)

NOTICE OF APPEAL

Notice is hereby given that Robert and Karen Joiner, Plaintiffs above named, hereby appeal to the United States Court of Appeals for the Eleventh Circuit from the Order dismissing this cause of action dated the 16th of September, 1994.

This 26th day of September 1994.

BURGE & WETTERMARK

By: /s/ Michael J. Warshauer
MICHAEL J. WARSHAUER
Georgia Bar Number 018720

2700 The Grand
75 Fourteenth Street, Northeast
Atlanta, Georgia 30309
(404) 875-2500

(Certificate of Service Omitted in Printing)

IN THE
UNITED STATES COURT OF APPEALS
FOR THE ELEVENTH CIRCUIT

No. 94-9131

ROBERT K. JOINER and KAREN P. JOINER,
Plaintiffs-Appellants,

v.

GENERAL ELECTRIC COMPANY,
WESTINGHOUSE ELECTRIC CORPORATION,
and MONSANTO COMPANY,
Defendants-Appellees.

On Appeal from the United States
District Court for the Northern District of Georgia
Honorable Orinda D. Evans, U.S.D.J.
Dist. Ct. No. 92-02137 1-CV-ODE

JOINT BRIEF OF DEFENDANTS-APPELLEES

February 20, 1995

* * * *

2. *Plaintiffs' Experts' Hypothesis Regarding PCBs is Without Scientific Support.*

Alternatively, the District Court found that, even if plaintiffs' experts "had not made unfounded assumptions about furans and dioxins, defendants still persuade the Court that plaintiffs' expert testimony would not be admissible." 864 F. Supp. at 1322. (R5-69-28) The basis for the Court's alternative ruling was Rule 702, which was construed in *Daubert* to require that the "subject of an expert's testimony must be 'scientific . . . knowledge' . . . which connotes more than subjective belief or unsupported speculation." 509 U.S. at ——, 113 S. Ct. at 2794-96, 125 L.Ed.2d at 480-81 (ellipsis in original; footnote omitted) (*quoted* below, 864 F.Supp. at 1326, R5-69-38-39). After careful consideration of the scientific authorities cited by the plaintiffs' experts, the Court concluded that their leap of faith from inadequate scientific bases to sweeping judgments about the causation of Mr. Joiner's small cell lung cancer constituted an unreliable methodology, and held: "The court need not address whether the studies that plaintiffs' experts rely upon were conducted in a scientific manner, for the studies simply do not support the experts' position that PCBs *more probably than not* promoted Joiner's lung cancer." *Id.* at 1326 (emphasis in original) (R5-69-39). This ruling was fully justified. *See O'Conner v. Commonwealth Edison Co.*, 13 F.3d 1090, 1106 (7th Cir. 1994), *cert. denied*, 114 S. Ct. 2711, 129 L.Ed.2d 838 (1994) (expert testimony not reasonably grounded in scientific literature must be excluded as speculative pursuant to *Daubert*); *Turbin v. Merrell Dow Pharmaceuticals*, 959 F.2d 1349, 1360 (6th Cir.), *cert. denied*, 113 S. Ct. 84, 121 L. Ed. 2d 47 (1992) ("[t]he analytical gap between the evidence and the inference to be drawn . . . is too wide") (cited with approval in *Daubert*, 509 U.S. at ——, 113 S. Ct. at 2798, 125 L. Ed. 2d at 484). The District Court criticized not the conclusions reached by plaintiffs' experts, but rather the methodology used to reach

them: unreliable speculation based upon inadequate grounds.

a. *Plaintiffs' Expert Testimony.*

Unquestionably, the most striking evidence supporting the District Court's conclusion that there is no scientific evidence that PCBs promote small-cell lung cancer in humans came from plaintiffs' own expert on the "mechanisms of carcinogenicity," who testified that there is no credible evidence as a scientific probability that PCBs promote small cell lung cancer in humans. Robertson Depo. at 14. Plaintiffs' other experts were also unpersuasive on the point. *See* Teitelbaum Depo. at 110 ("there is no such study"); Schecter Depo. at 68-69 (admitting his opinions are based solely on extrapolations from animal studies). In summary, plaintiffs' experts all agreed that there are no epidemiological studies which conclude that PCBs cause or promote small cell lung cancer in humans. *See* 864 F. Supp. at 1324, n.25. (R5-69-33, n.25) However, the court below did not require epidemiological proof as a matter of law.

b. *Two Studies With Infant, Suckling Mice.*

Instead, the District Court devoted several pages of its thorough opinion to a detailed review of the two experimental *animal* studies cited by the plaintiffs' experts as the scientific underpinning for their hypothesis that PCBs could promote small cell lung cancer in human beings—studies which involved tests on infant, suckling mice first dosed with known carcinogens, then with huge quantities of PCBs (*not* mineral oil contaminated with low levels of PCBs) injected directly into the body cavity containing the most vital organs. 864 F. Supp. at 1322-1326. (R5-69-28-39) The District Court concluded:

> Dr. Robertson admitted that his opinion that PCBs are "promoting agents" is based on only two studies in infant, suckling mice (Robertson Depo., at 14-15). . . . [A]s to those two studies, Dr. Robertson admitted that (1) the lung cancer promoted in the infant mice was not identified as small cell carcinoma (Robertson Depo., at p. 15), (2) he did not know whether the same effect had been produced in adult mice (Robertson Depo., at p. 23), (3) the tumors produced were dose-dependent (Robertson Depo., at p. 26), and (4) after having been administered a known initiating carcinogen, the mice were dosed with 100% PCBs by either injecting it directly into the peritoneum (the body cavity containing the most vital organs) or directly into the stomach (Robertson Depo., at p. 27-28).
>
> A review of the two studies themselves reveals that [the] infant mice developed alveologenic adenomas, not small cell carcinomas (Robertson Depo., Exhibits 2 & 3).

864 F. Supp. at 1322-23 (footnotes omitted). (R5-69-29-30) The District Court further found that "[b]oth Dr. Schecter and Dr. Teitelbaum [also] relied upon the [two] mice studies in opining that PCBs promoted Joiner's lung cancer." *Id.* at 1323 (citing Schecter Depo. at pp. 66-69; Teitelbaum Depo. at p. 48, 78, 81). (R5-69-30) There is no animal study showing lung cancer was promoted in any other species. (Teitelbaum Depo. at 87.)

The District Court held that plaintiffs' reliance on the two infant suckling mice studies was unsound, and revealed that their opinions did not constitute "scientific knowledge," because: (1) there are only two studies (864 F. Supp. at 1322-23) (R5-69-28-32); (2) "the studies obviously used massive doses of PCBs" (*id.*); and (3) Dr. Teitelbaum admitted the mice studies' findings were "preliminary" (*Id.*). Given the nature and limitations of the particular animal studies relied upon by the plaintiffs' experts, the District Court concluded that those studies provided no scientific basis for the opinion prof-

fered by the plaintiffs' experts that PCBs promote small cell lung cancer in human beings.[20]

On this record, the Court concluded that "defendants have sufficiently called into question the validity of plaintiffs' experts' reliance on the mice studies," and held: "Therefore, the burden shifts to plaintiffs to demonstrate by a preponderance of proof that their experts' opinions are admissible." *Id.* at p. 1324 (R5-69-32); *see also* pp. 10-11, *supra*. The Court then gave plaintiffs every opportunity to make such a showing.

Plaintiffs, however, chose "to proceed as if the only issue is whether animal studies *ever* can be a proper foundation for an expert's opinion . . . that is not defendants' argument." *Id.* (emphasis added). Plaintiffs offered nothing more. While the district court properly analyzed plaintiffs' experts' methodology with reference to the specific bases offered as the underpinnings of plaintiffs' experts' proposed opinions in this case, plaintiffs' arguments sought a referendum on the probative value of animal studies in the abstract. The District Court, finding plaintiffs' argument "unresponsive to the issue at hand," ruled that plaintiffs' experts' methodology and opinions were flawed because they "erred in relying on the mice studies to opine that PCBs caused Joiner's lung cancer to a reasonable degree of medical certainty." *Id.* (citations omitted). The studies were too few, too tenuous in application to the present case, and simply inadequate as a basis for the ex-

---

[20] Dr. Teitelbaum testified that in the two studies with infant mice an increase in tumors was observed *only* at the highest dose. (Teitelbaum Depo. Exh. 12-C.) The authors of the studies also recognized the limitations of using infant mice, writing: "further experiments are required to determine whether promotion by PCBs . . . is a special property of tumors initiated soon after birth." In the second study, the authors reported that "exposure also to PCB resulted in a lower tumor incidence in some of the treatment groups" and that "this *protective action was most evident for lung tumors* in both sexes at 18 months of age." (Robertson Depo., Exhibit 2; emphasis added).

perts' opinions, the Court ruled. "The analytical gap between the evidence presented and the inference to be drawn . . . is too wide," the Court wrote. 864 F. Supp. at 1323 (citation omitted). (R5-69-31)

c. *"Epidemiological" Studies Cited by the Joiners' Counsel.*

In Plaintiffs' Opposition to Defendants' Motion, their counsel purported to identify epidemiological studies which they claimed supported their experts' opinions. In discussing these studies, the district court noted with some irony that all three of plaintiffs' experts testified in their depositions that there are no studies showing PCBs cause, contribute to, or promote small cell lung cancer in humans. 864 F. Supp. at 1324, n.25. (R5-69-33, n.25) Still, the District Court carefully considered each of the studies cited by plaintiffs' counsel, and made detailed findings as to why none provided a basis for reaching the conclusion proposed by plaintiffs' experts. The Court found that two studies cited by the plaintiffs reported no statistically significant relationship between PCBs and lung cancer, one revealed "no ground" for any such link, and still another "never mentions PCBs." 864 F. Supp. at 1324-26 (R5-69-33-40) Another showed causation not by PCBs, but by another chemical; even plaintiffs' expert Dr. Teitelbaum called it "not very convincing." *Id.* at 1326 (R5-69-38) (citing Teitelbaum Depo. at 89-90). Based upon this thorough analysis, the District Court concluded: "in every case defendants show that the studies [cited by the plaintiffs] are either equivocal or not helpful. . . ." *Id.* at 1324. (R5-69-34)

The District Court therefore held the plaintiffs' experts' *methodology* inadequate, because there was no scientific link between the cited studies and the proffered opinions:

> [T]he Court is not persuaded by a preponderance of proof that the studies support the "knowledge" the experts purport to have (*i.e.*, that PCBs, "to a 'rea-

> sonable degree of medical certainty,'" *Wells*, 615 F. Supp. at 295, promote small cell lung cancer in humans). *See Turpin*, 959 F.2d at 1360. ("The analytical gap between the evidence presented and the inference to be drawn on the ultimate issue of human birth defects is too wide."); *cf. Wells*, 788 F.2d at 745. ("[T]he basic *methodology* employed to reach . . . a conclusion [must be] sound.")

Id. at 1326 (ellipsis and brackets in original, emphasis supplied). The cases confirm that a district court applying the *Daubert* analysis must exclude proffered expert opinion testimony as methodologically flawed when it is founded upon unreliable bases. In *McLendon v. Georgia Kaolin Co.*, 841 F. Supp. 415 (M.D. Ga. 1994), for example, a District Court in this Circuit applying the *Daubert* analysis stated recently: "The court must make the ultimate determination of whether a reliable basis underlies the expert opinion." *Id.* at 418 (Owens, C.J.) (citing *Smith v. Ortho Pharmaceutical Corp.*, 770 F. Supp. 1561, 1573 (N.D. Ga. 1991)).

The Joiners contend that the District Court improperly rejected the *conclusions* of plaintiffs' experts, rather than the *methodology* used to reach those conclusions. (Appellants' Brief at 14.)[21] This argument ignores what the

[21] The Joiners assert that the District Court opinion states defendants have attacked plaintiffs' experts' "conclusions." (*Id.*) In fact, the District Court wrote: "Defendants . . . attack[] the conclusions that Plaintiffs' experts draw *from the studies they cite.*" 864 F. Supp. at 1322 (emphasis supplied) (R5-69-28). Thus, even the portion of the Court's Order cited by the plaintiffs reflects the District Judge's emphasis on the invalidity of the *leap of faith* from the cited bases for plaintiffs' experts' conclusion to their opinions, rather than the conclusions themselves. As the Third Circuit stated recently in *In re Paoli Railway Yard PCB Litigation*, 35 F.3d 717, 746 (3d Cir. 1994), *reh'g denied*, Oct. 14, 1994, *petition for cert. filed*, Dec. 16, 1994:

> When a judge disagrees with the conclusions of an expert, it will generally be because he or she thinks that there is a mistake at some step in the investigative or reasoning process of that expert. . . This is especially true given that the expert's view that a particular conclusion "fits" a particular case must itself constitute scientific knowledge—a challenge to "fit" is very close to a challenge to the expert's ultimate conclusion about the particular case, and yet it is part of the judge's admissibility calculus under *Daubert*.

Id. at 746 (emphasis in original; footnotes omitted).

Court did. Simply put, the District Court did not reject the conclusions of plaintifls' experts: rather, the District Court analyzed the proffered *bases* for those opinions and correctly concluded that those bases do not constitute scientific support for those opinions. The court looked to (1) the record facts upon which plaintiffs' experts said they relied, (2) the animal studies upon which their counsel said they relied and (3) the epidemiological studies upon which they said they relied, in order to determine whether those facts and studies suported the opinions. It was, in short, the plaintiffs' experts' "methodology" of assuming unproven facts, relying upon limited and questionable animal research, and citing unsupportive epidemiological studies to leap freely to sweeping opinions about causation that the District Court found unscientific and inadmissible.

Because plaintiffs' experts performed no independent research to verify their proffered opinions, reliance upon this leap of faith from inapposite facts and literature *is* their methodology, for they did no more. A methodology which generates opinions based upon inapposite scientific literature is flawed. The District Court properly concluded that, under *Daubert*, because they are without scientific support, "the opinions of plaintiffs' experts do not rise above 'subjective belief or unsupported speculation.'" 864 F. Supp. at 1326 (footnote omitted). (R5-69-39) Plaintiffs' protestations to the contrary notwithstanding, this is a perfectly appropriate basis to exclude the evidence.

Daubert also identifies whether an hypothesis has been tested as the "key question" in determining whether it is derived by the scientific method or is instead speculative and inadmissible. 509 U.S. at ——, 113 S. Ct. at 2796, 125 L. Ed. 2d at 482-83. The plaintiffs' experts' hypothesis *has* been tested and is without scientific support. None of the epidemiological studies support a link between exposure to PCBs and lung cancer in humans, let alone Mr. Joiner's small cell cancer of the lung.[22] Moreover, the epidemiological studies refute the Joiners' hypothesis that PCBs "promote" lung cancer "initiated" by cigarette smoking.[23] In an effort to obscure this fact, plaintiffs' expert Dr. Teitelbaum suggested that the Joiners' hypothesis is "an impossible research question to investigate. . . ." (Teitelbaum Depo. at 110-111.) But,

> "Scientific" knowledge is generated through the scientific method—subjecting testable hypotheses to the crucible of experiment in an effort to disprove them. An opinion that defies testing, however defensible or deeply held, is not scientific.

U.S. v. Bynum, 3 F.3d 769 (4th Cir. 1993), *cert. denied*, 114 S. Ct. 1105, 127 L. Ed. 2d 416 (1994) (citing *Daubert's* emphasis on the testability of proffered expert hypotheses). Indeed, one of the sources cited by the Supreme Court in *Daubert* put the matter very succinctly: "[t]he statements constituting a scientific explanation must be capable of empirical test." *Daubert*, 509 U.S. at ——, 113 S. Ct. at 2796-97, 125 L. Ed. 2d at 483 (quoting C.

---

[22] When all of the epidemiological studies of workers exposed to PCBs (*not* contaminated mineral oil transformers as in this case) are taken together, "you have a body of evidence that doesn't give the slightest inkling that these things [PCBs] cause human cancer." (Cole 10/21/93 Depo., R, Exh. filed 2/2/94 (hereinafter "Cole Depo."), at 88.)

[23] "[If PCBs were assumed to promote lung cancer initiated by cigarette smoking] we would expect some very consequential increase in lung cancer rates. . . . But that hasn't happened." (Cole Depo. at 115, 116).

Hempel, *Philosophy of Natural Science* 49 (1966)). Because the plaintiffs' experts' hypothesis is based upon unsupported premises, is not consistent with the methodology of science, and thus does not constitute "scientific knowledge," the District Court properly excluded the plaintiffs' experts' opinions pursuant to *Daubert*. The District Court's exclusion of plaintiffs' experts' speculative opinions should stand.

* * * *

SUPREME COURT OF THE UNITED STATES

No. 96-188

GENERAL ELECTRIC COMPANY, *et al.*,
Petitioners

v.

ROBERT K. JOINER, *et ux.*

ORDER ALLOWING CERTIORARI

Filed March 17, 1997

The petition herein for a writ of certiorari to the United States Court of Appeals for the Eleventh Circuit is granted.

March 17, 1997